ARCHIVES OF OTOLARYNGOLOGY

VOLUME 43 FEBRUARY 1946 NUMBER 2

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TESTING THE HEARING OF YOUNG CHILDREN

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WHEN members of the family suspect that a baby is deaf or hard of hearing, they are of course alarmed. They want a diagnosis as early as possible, and this is one of the reasons why it is desirable to examine the child with defective hearing as early in its life as possible.

However, there are also other reasons why it is expedient to recognize the presence and to determine the seriousness of a hearing defect early. Normal development of speech depends on whether enough acoustic stimuli reach the acoustic centers and the Wernicke center to train them. Only when these centers are charged often enough (literally innumerable times) with those stimuli, will the motor speech centers be stimulated in turn by them. The latter stimulation is synonymous with the impetus necessary for the development of spontaneous speech.

If an examination of the hearing discloses that the distance at which a child hears is reduced, one will naturally see to it that the child is subjected to stimuli within the distance at which it can hear.

If a complete lack of hearing for a few, though not for all, tones is discovered, there is still hope that systematic training of the hearing organs will set in motion those impulses necessary to normal development of speech. Another reason for early diagnosis and treatment is the fact that nature has reserved the first two years of life for the acquisition of spontaneous speech. It has been observed that many children who during these years suffer from enfeebling illnesses cannot start to speak without outside aid, even if those illnesses had nothing to do with either the hearing or the speech organs.

A method of examining the hearing of very young children is therefore extremely important.

In recent years audiometry has replaced some methods of testing the acuity of hearing almost exclusively. Its superiority over the use of tuning forks or whistles lies chiefly in the pureness and in the measurable loudness of the audiometric tones. Its application, however, calls for active cooperation on the part of the patient, who is requested to answer deliberately whether each single tone can be heard or not, a fact which renders the use of the audiometer undesirable with very young children and infants.

Hughson and Thompson [1] recently stated that "reasonably accurate audiograms can be made on children from the age of two years and upward." They also stated that "the reaction of a child with impaired hearing manifests itself at some level between 20 and 60 decibels above threshold." While Westlake [2] concluded from his own experience: "Children of three and four years of age show less consistency in their responses to the pure-tone tests and show a wider deviation from the accepted normal threshold than older ones do, but these are very probably due to other factors than auditory acuity."

This conclusion is in accord with my own experience in the study of children with impaired hearing. I have seen audiometer tests made on children up to 8 years of age which indicated complete deafness although the same children showed positive reactions to tones introduced directly into their ears.

The apparatus for the "direct tone introduction method" is a set of whistles (see accompanying figure) from Urbantschitsch's organon,

One of the whistles used to introduce tones directly into the young child's ear.

comprising a tone range from F 1 to f 4. The whistles of the big contra-octave are not practical, probably, because their tongues are too heavy to be moved by a short blow.[3] This, however, is of no importance in the development of speech [4] since even the bass voice in speaking does not comprehend tones lower than A.[4]

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1. Hughson, W., and Thompson, E.: Audiometry in the Diagnosis and Treatment of Deafness in Children, Ann. Otol., Rhin. & Laryng. **54**:480, 1944.

2. Westlake, H.: Hearing Acuity in Young Children, J. Speech Disorders **7**:1, 1942.

3. It is essential for the successful application of the test to give a short vigorous blow (about one second) rather than a long, persistent one. The sudden onset of the stimulus releases reflexes different from those caused by the slow, soft onset—a difference in reaction which may be noticed in various other reflexes.

4. In testing infants, the distance at which tones can be heard cannot be determined. It is comparatively easy, however, to teach older children to indicate when and at what distance they hear the tone.

For the direct introduction of a tone, a rubber tube to which an "ear olive" is attached is inserted into the external auditory meatus, while the other end of the tube touches the opening of the whistle through which the tone escapes. Thus the tone is delivered directly into the ear. It is advisable to cover the ear olive with fine rubber in order to prevent the air stream from entering the ear. Some subjects react only positively if the olive is uncovered. The reflexes expected as a positive answer to the direct introduction of a tone are blinking, grimaces and incidentally the "eye muscle reflex."

With regard to the "eye muscle reflex" I refer to two publications.[5] At this point I want to give only a brief description of the reflex and of the way it was discovered. By the method described, the tones of the whistles were introduced into the external auditory meatus by an uncovered ear olive. At the sounding of a tone some patients showed more or less intense swaying movements. On closer examination of the eyes in the cases reported,[5] I noted at each resounding of the tone and resulting swaying of the body a horizontal and occasionally a vertical jerking of the eyeballs in a direction away from the source of the sound, followed by an accelerated movement back to the initial position. The first patient in whom I found the jerking of the eyeball was a hard of hearing girl, 5 years of age. When she was told to turn her eyeballs toward the "charged" ear, the jerking of the eyeballs was as strong as when they had been in medial position. While all tones ranging from C to a3 produced this phenomenon, tones below and above this range remained ineffective. A change in the position of the head did not influence the horizontal movement from one side to the other. The blowing of air into the meatus also failed to produce the sypmtom. The results of the test of the other ear were similar. However, when the tone was produced with the whistle held near the ear instead of the tone being introduced directly into the meatus by means of the otoscope, no matter how close the proximity of the whistle, no movements of the eyeballs, disturbances of the equilibrium or auropalpebral reflexes (blinking) could be observed. In this case no vestibular sensitivity to turning or to cold or warm rinses of the ear was apparent.

I have referred the reader to the original publications.[5] However, I wish to mention two important facts. In over a hundred persons examined, whenever there was the ocular jerk the patient, without a single exception, stated that he had heard the tone. Moreover, none of the patients showed the symptom of fistula on compression of air, or aspiration of air from, the external auditory canal—a symptom with which the one described is by no means identical.

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5. Froeschels, E.: Ueber einen durch Schallreiz ausgelösten Augenmuskelreflex bei Taubstummen, Monatschr. f. Ohrenh. **60**:281, 1926; **61**:776, 1927.

Since one is concerned here with reflexes, the method can be employed without depending on the cooperation of the patient, a fact which with young children makes it more reliable than the audiometric test, which requires the full attention as well as the cooperation of the examined person. It stands to reason that the whistle test does not indicate the single notes which can be heard. A positive reaction may be the response to overtones of the single whistle. But if there is a positive reaction to different whistles, one may safely assume that different tones have been heard, especially since different pipes comprise different overtones and some do not contain any of the overtones appearing in others. If there is a positive answer to almost all or to all the whistles, hearing throughout the whole range of the whistles is probably present. Experience during the treatment has proved this assumption to be right.

Among the hundreds of children whom I have examined since I started using the direct tone introduction method there has been no child under the age of 3 until a few months ago, when a girl of 15 months was brought to me. Her mother suspected that the infant was deaf because she could detect no response to speech, tones or noises; but no exact proof of this assumption of a hearing defect could be given. Otologists who had examined the child found the drum membranes normal and informed the mother that further examination had to be postponed several years, until the child was old enough to be tested.

The height and the weight of the patient were normal. There were no pathologic conditions except those concerning the hearing. There had been no previous diseases. The child behaved intelligently and in playing showed no deviation from normalcy. The family history was normal except that rubella had developed in the mother during the third month of pregnancy. Such an occurrence of this disease has taken on considerable significance in recent years.[6]

I familiarized the child with the whistle by putting it into my ear and then, playfully, on her body, on her nose and, taking her unawares, into her left ear. At the same instant I blew into the whistle. I repeated this with all the whistles and each time obtained positive

6. Altmann, F., and Dingmann, A.: Congenital Deafness and Cataract Following Rubella in the Mother, Arch. Otolaryng. **42**:51 (July) 1945. Erickson, C. A.: Rubella Early in Pregnancy Causing Malformation of Eyes and Heart, J. Pediat. **25**:281, 1944. Gregg, N. M.: Congenital Cataract Following German Measles in the Mother, Tr. Ophth. Soc. Australia **3**:35, 1941. Reese, A. B.: Congenital Cataract Following German Measles in the Mother, Am. J. Ophth. **27**:483, 1944. Swan, C., and others: Congenital Defects in Infants Following Infectious Diseases During Pregnancy, M. J. Australia **2**:201, 1943. Swan, C.; Tostevin, A. L.; Mayo, H., and Black, G. H. B.: Further Observations on Congenital Defects in Infants Following Infectious Diseases During Pregnancy, with Special Reference to Rubella, ibid. **1**:409, 1944.

responses on the left side and occasionally also on the right side. These reactions were blinking, occasional grimaces and smiling. Five examinations gave the following results (+, positive answer; 0, no answer):

Right		Left	Right		Left
+	C	+	0	a 1	+ ¼ m; (whistle tried on distance)
	D	Whistle did not function			
0	E	+	+	h 1	+
0	F	+	+	c 2	+
0	G	+	+	d 2	+
+	A	+	+	e 2	+
0	H	+	+	f 2	+
+	c	+	+	g 2	+
+	d	+	+	a 2	+
+	e	+	+	h 2	+
+	f	+	0	c 3	+
+	g	+	+	d 3	+
0	a	+	+	e 3	+
+	h	+	+	f 3	+
+	c 1	+	0	g 3	+
0	d 1	+	+	a 3	+
+	e 1	+	+	h 3	+
+	f 1	+ First smiling	+	c 4	+
0	g 1	+	0	d 4	+
			+	e 4	+

Having completed the examination, I suggested the use of a hearing tube to the end of which a funnel is attached. I advised the mother to speak vowels, syllables and words into the funnel and to use as further stimuli music, as well as various noises—a procedure to be repeated frequently, about a hundred times a day for a minute each time. The purpose of this treatment is the stimulation and the training of the acoustic and the Wernicke center as well as the auditory music center (second temporal convolution) in receiving and distinguishing sounds, which in this child, contrary to the child of normal acuity of hearing, would not be received otherwise.

It is my intention later to supply the child with ear phones and to have her listen to radio programs repeatedly. Considering the great number of infants in this country who are afflicted with defects of hearing, I submit that special radio programs should be arranged by persons expert in the field of hearing and speech; they could render inestimable service in promoting spontaneous development of speech in these children.

SUMMARY

In summarizing I wish to repeat that it is essential for the development of speech of the hard of hearing child to examine the hearing early in the life of the child. As the audiometric method is not practicable when applied to a very young child, the method of direct introduction of tones should be used, even though the findings are not as exact as those of audiometry. If hearing throughout the full range of

the whistles or at least throughout a considerable part of that range can be detected, stimuli should be introduced into the ear of the child with the help of a hearing tube. The stimuli should consist of musical sounds, noises, single speech sounds, single and, later on, combined syllables, monosyllabic words and, at a more advanced stage, words containing two and more syllables, increasingly longer phrases, and so on—in other words, a system of sounds imitating the sounds and noises reaching a child of normal hearing.

It is my conviction that spontaneous development of speech could be brought about in at least a number of hard of hearing children if this method, which offers a system where nature fails, were employed.

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TREATMENT OF ACUTE PHARYNGEAL INFECTIONS BY INSUFFLATION OF SULFANILAMIDE POWDER

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THE standard treatment of acute pharyngeal infections—frequent irrigations with hot saline solution in cases of acute tonsillitis or pharyngitis, and hydrogen peroxide gargles and applications of sodium perborate in cases of ulcerative tonsillitis associated with Vincent's micro-organisms—often presents problems in nursing and administration. In view of the high incidence of pharyngeal infections among military personnel, therapy that offers simplification of management and improvement of results in comparison with the methods in common use deserves consideration.

Acute pharyngeal and tonsillar infections are inflammatory processes largely confined to surfaces. Accordingly, it appeared worth while to us to treat a series of patients for these conditions by local applications of sulfanilamide powder. It was our intention to determine whether such therapy offered greater advantages for military personnel than the standard remedies.

In discussing the local use of sulfonamide compounds, Spink [1] stated:

> The chief virtue of local therapy is that it provides much higher tissue concentrations of the drug in a relatively small area in a shorter period of time than can be obtained when the drugs are administered orally or parenterally. Such a procedure also avoids unpleasant systemic reactions.

Although there have been many instances in the literature of the use of solutions of sulfonamide drugs in the treatment of patients with acute infections of the upper respiratory tract, reports dealing with the local application of the powdered form of sulfonamide compounds to infected throats have been sparse. Freeman [2] reported the results in a series of 34 patients similar to ours treated by the topical application of sulfathiazole powder. He concluded that the method rapidly produced subjective and objective relief and materially shortened the course of the infections. Fenton [3] also claimed favorable results from the treat-

1. Spink, W. W.: Sulfanilamide and Related Compounds in General Practice, Chicago, The Year Book Publishers, Inc., 1943, chap. 10, p. 204.

2. Freeman, M. S.: Local Use of Sulfathiazole Powder for Acute Pharyngeal Infections, Arch. Otolaryng. **37**:496-501 (April) 1943.

(Footnotes continued on next page)

ment of pharyngeal infections with sulfathiazole powder with the exception of the ulcerations of Vincent's angina. Linton [4] successfully treated 4 patients with Vincent's angina of the tonsils by having them dissolve sulfathiazole tablets on the tongue frequently.

This report consists of our findings in the first 69 patients treated for acute tonsillitis, Vincent's ulcerative tonsillitis and acute pharyngitis by the local insufflation of sulfanilamide powder.[5] A series of 54 controls is included. The sulfanilamide powder was insufflated with either an air-compressed or a hand-compressed De Vilbiss glass powder blower once a day in the early part of the series and twice a day in the latter part. The patients were instructed to indulge in as little expectoration as possible for at least two hours after each treatment and to ingest nothing during the same period. Examinations showed that the drug was present in the throat as long as three hours after insufflation, particularly in the cases of ulcerative tonsillitis. In the cases of acute pharyngitis the drug was retained in the throat for only a short while.

ACUTE TONSILLITIS

In this group there were 32 patients treated locally by insufflations of sulfanilamide powder and 32 controls. The treatment of the latter consisted of irrigations of the throat with hot saline solution. All except 4 of the patients in the control series were observed and treated by one of us (W. B. K.). The average time spent in the hospital by the sulfanilamide-treated group before clinical cure was achieved was 4.4 days, the average number of insufflations for each patient was 4.4 and the average length of time before soreness and dysphagia disappeared was 3.3. In comparison, in the controls clinical cure was effected in 6.0 days and local symptoms ceased in 4.5 days.

The patients in this series were suffering from sore throat and dysphagia, mild systemic symptoms and a fever, with temperatures between 99 and 102 F. Their tonsils were swollen, hyperemic and covered with varying amounts of exudate. Both tonsils were most commonly involved. Patients who presented the clinical appearance of severe toxicity with prostration and temperatures above 102 F. were treated by oral administration of sulfadiazine. We encountered 6 such patients, and all showed a favorable and prompt response to this therapy.

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3. Fenton, C. A.: Local Use of Sulfathiazole in Otolaryngologic Practice, Arch. Otolaryng. **37**:491-495 (April) 1943.

4. Linton, C. S.: The Treatment of Vincent's Angina of the Tonsil: Preliminary Report, J. A. M. A. **123**:341 (Oct. 9) 1943.

5. Over 150 patients had been treated by this method when this paper was submitted for publication. The therapeutic results continued to be as satisfactory as is indicated in this report.

Blood sulfanilamide levels were determined for 8 patients in this group two hours after the administration of sulfanilamide powder, and in no instance could sulfanilamide be detected in the blood.

In view of the fact that a number of patients were treated from the outpatient department, complete bacteriologic investigations were made for only 71 per cent. Material from the throats of the patients treated with sulfanilamide powder in the wards was cultured before the first treatment; then material for another culture was taken after the infection subsided to see if there was any change in the flora of the tonsils. Similar cultures were also made for the control patients. The following micro-organisms when present in small numbers, singly or in combination, were considered as normal flora of the tonsils: Neisseria catarrhalis, Staphylococcus aureus, Staphylococcus albus, Streptococcus viridans (alpha hemolytic and alpha prime), pneumococci, Haemophilus influenzae and diphtheroids. If, however, any of these micro-organisms appeared in large numbers, singly or in combination, they were considered pathogenic. In this study, Streptococcus haemolyticus (beta) was the principal micro-organism found in the infected tonsils. It was present in the tonsils of 15 of the 32 sulfanilamide-treated patients and in 16 of the 32 controls. After clinical cure had been obtained, cultures of material from the tonsils failed to show beta hemolytic streptococci in 6 of the 15 patients in the sulfanilamide group, while of the 16 in the control group, 3 were shown to be free of this micro-organism. Str. viridans was found in large numbers in the tonsils of 3 patients of the sulfanilamide-treated group and in those of 3 of the control group. The action of sulfanilamide on this micro-organism was inconclusive. Staph. aureus was isolated 3 times in the sulfanilamide-treated group and the control group. Neither form of treatment caused the disappearance of this micro-organism (tables 1 and 2).

VINCENT'S ULCERATIVE TONSILLITIS

The patients considered in this group were those in whom the ulceration involved only the tonsil (or tonsils). All had unilateral involvement at first, but bilateral lesions developed in several before the completion of treatment. Twenty-eight patients with Vincent's ulcerative tonsillitis were treated by insufflations of sulfanilamide, while 23 were used as controls. Fifteen of the controls did not come under our direct observation; the data presented on them were obtained from the hospital charts.

For the patients treated by insufflations of sulfanilamide the average length of time before clinical cure was attained was seven days, the average number of treatments was 8.9 and the average number of days required for pain to disappear from the infected tonsil was 4.7. In con-

trast, among the controls, an average of 8.7 days was required for the infection to clear and 6.4 days for the pain to disappear from the infected area. In considering these averages, it should be noted that

TABLE 1.—*Patients Suffering from Acute Tonsillitis Who Were Treated with Sulfanilamide Powder*

Patient	Days of Treatment	Treatments	Days Before Cessation of Pain	Bacteriologic Findings: Before Treatment	Bacteriologic Findings: After Treatment	Comment
1	2	3	2	Str. haemolyticus	Staph. albus	
2	2	4	2	Normal	Normal	
3	6	4	5	Str. viridans	Staph. albus, pneumococcus	
4	9	9	6	Str. haemolyticus, Str. viridans	Staph. aureus haemolyticus	Blood sulfanilamide level zero
5	2	2	1	Str. haemolyticus	.....	
6	3	3	2	Str. haemolyticus	Normal	
7	6	6	6	Normal	Normal	Blood sulfanilamide level zero
8	10	10	8	Normal	Normal	Blood sulfanilamide level zero
9	3	3	2	Normal	Normal	
10	5	5	4	Str. haemolyticus	Normal	
11	3	3	3	Normal	.....	
12	5	5	3	Str. haemolyticus	Str. viridans	Blood sulfanilamide level zero
13	3	2	2	Normal	Normal	Blood culture negative
14	3	3	3	Str. haemolyticus	Str. haemolyticus, Staph. aureus	
15	4	4	4	Str. haemolyticus, Staph. aureus	Str. haemolyticus, Staph. aureus Str. viridans	
16	4	4	4	Str. haemolyticus	Str. haemolyticus	
17	3	4	2	Normal	Str. haemolyticus	Blood sulfanilamide level zero
18	4	5	3	Str. haemolyticus	Normal	
19	3	5	3	Str. haemolyticus	Str. haemolyticus	Blood sulfanilamide level zero
20	3	6	3	Normal	Normal	Blood sulfanilamide level zero
21	3	3	2	Str. viridans, Staph. albus, pneumococcus	.....	
22	5	4	4	Normal	.....	
23	4	4	3	Normal	.....	
24	4	3	3	Str. haemolyticus	.....	
25	6	5	4	Str. haemolyticus, Staph. aureus haemolyticus	.....	
26	3	6	3	Staph. aureus	.....	
27	5	4	4	.....	.....	
28	6	6	3	Str. haemolyticus	Str. haemolyticus	
29	6	7	3	Str. haemolyticus	.....	Blood culture negative
30	6	6	3	.....	.....	
31	6	8	4	Normal	Str. viridans	
32	3	4	2	.....	.....	Blood sulfanilamide level zero
Average	4.4	4.4	3.3			

among the patients treated by insufflations of sulfanilamide there were 3 whose period of treatment was of unusually long duration. In 2 instances this was caused by activation of an ulcer in an opposite tonsil

coincident with the subsidence of ulceration in the first tonsil. In the third instance there was massive bilateral involvement which persisted on one side a long time after the other side had healed. If the time required for the treatment of the tonsils which became secondarily infected is eliminated, the average number of days of treatment for the sulfanilamide group becomes 5.6, as compared with 8.7 for the control group.

TABLE 2.—*Control Patients with Acute Tonsillitis*

Patient	Days of Treatment	Days Before Cessation of pain	Bacteriologic Findings		Comment
			Before Treatment	After Treatment	
1	5	3	Str. viridans	Str. viridans, Staph. aureus	
2	8	3	Str. haemolyticus	Normal	
3	4	4	Normal	Normal	
4	5	4	Str. haemolyticus	.....	
5	8	5	Str. haemolyticus	Str. haemolyticus	
6	5	3	Normal	Str. viridans	
7	4	4	Normal	.....	
8	5	4	Str. haemolyticus	Str. haemolyticus	
9	3	3	Normal	Normal	
10	8	7	Str. haemolyticus	.....	Blood culture negative
11	9	8	Str. haemolyticus	Normal	
12	5	4	Str. haemolyticus	Str. haemolyticus	
13	4	3	Str. haemolyticus, Str. viridans	Str. haemolyticus	
14	5	4	Normal	Str. viridans	
15	4	3	Str. haemolyticus	Str. haemolyticus	
16	5	2	Str. haemolyticus	.....	
17	6	3	Normal	Str. viridans, Staph. albus	
18	7	4	Str. haemolyticus, Staph. aureus	Str. viridans	
19	9	6	Str. haemolyticus	.....	
20	6	4	.....	.....	
21	5	4	Str. haemolyticus	.....	
22	6	4	Str. viridans	.....	
23	6	5	Str. haemolyticus	.....	
24	6	5	Normal	.....	
25	7	5	Normal	.....	
26	7	4	Normal	.....	
27	7	4	Str. haemolyticus, Staph. aureus	Str. haemolyticus, Staph. aureus	
28	10	8	Str. haemolyticus	Str. haemolyticus	
29	4	4	.....	.....	
30	7	5	Staph. aureus	.....	
31	7	5	Normal	.....	
32	6	5	Normal	.....	
Average	6.0	4.5			

Most of the patients for whom bacteriologic cultures were not made were patients treated in the outpatient clinic.

In dealing with the ulcerative lesions, the necrotic membrane and tissue formed in the depths of the ulcer were removed with a cotton applicator before the sulfanilamide powder was insufflated, which permitted the powder to settle on a raw surface. In the control group

TABLE 3.—*Patients Suffering from Vincent's Ulcerative Tonsillitis Who Were Treated with Sulfanilamide Powder*

Patient	Days of Treatment	Treatments	Days Before Cessation of Pain	Bacteriologic Findings in				Comment
				Smear Before Treatment	Culture Before Treatment	Smear After Treatment	Culture After Treatment	
1	5	5	4	Fusiform bacilli and spirilla	Normal	0	.....	
2	2	2	1	Fusiform bacilli and spirilla	Str. viridans	0	Str. haemolyticus	
3	6	6	5	Fusiform bacilli and spirilla	Normal	.....	Normal	Blood sulfanilamide level zero
4	8	8	5	Fusiform bacilli and spirilla	Str. viridans	Fusiform bacilli and spirilla	Normal	Recurrence in right tonsil in 3 weeks; yielded to powder within 4 days; blood sulfanilamide level zero
5	3	3	2	Fusiform bacilli and spirilla	Str. viridans	0	Str. viridans	
6	6	6	6	Fusiform bacilli and spirilla	Str. haemolyticus	Fusiform bacilli and spirilla	Str. haemolyticus, Str. viridans	
7	16	16	15	Fusiform bacilli and spirilla	Str. haemolyticus	Fusiform bacilli and spirilla	Normal	Ulceration only on right side for 7 days; then left broke down—9 more days; recurrence on left in 3 weeks
8	8	8	7	Fusiform bacilli	Normal	0	Str. haemolyticus	Involvement of gum
9	2	2	2	Fusiform bacilli and spirilla	Str. viridans	Fusiform bacilli and spirilla	Str. viridans	
10	11	17	4	Fusiform bacilli and spirilla	Str. viridans, Staph. aureus	0	Normal	Blood sulfanilamide level zero
11	5	5	4	Fusiform bacilli and spirilla	Normal	Fusiform bacilli and spirilla	Normal	
12	5	5	3	.....	.....	.....	Normal	
13	3	5	1	Fusiform bacilli and spirilla	Str. haemolyticus, Staph. albus	.....	.....	
14	3	6	3	.....	Staph, aureus, pneumococcus	.....	.....	

15	10	20	4	Fusiform bacilli	Str. haemolyticus	Fusiform bacilli and spirilla	Str. haemolyticus, Str. viridans	Patient returned with massive involvement on left 3 days after discharge; blood sulfanilamide level zero
16	4	8	3	Fusiform bacilli and spirilla	Str. haemolyticus, Str. viridans	.....	.....	
17	21	36	14	Fusiform bacilli and spirilla	Staph. aureus, Str. haemolyticus	0	Normal	Right tonsil healed in 10 days, then left became involved; it healed in 11 days; massive involvement of both sides
18	5	5	0	Fusiform bacilli and spirilla	.....	.....	.....	Involvement of gum; routine measures tried for 17 days at first
19	4	3	3	Fusiform bacilli and spirilla	.....	.....	.....	
20	6	8	5	Fusiform bacilli and spirilla	Normal	0	.....	Involvement of gum
21	4	4	3	Fusiform bacilli and spirilla	.....	.....	.....	
22	4	5	3	Fusiform bacilli and spirilla	.....	.....	.....	
23	7	8	5	Fusiform bacilli and spirilla	Normal	0	Normal	Blood sulfanilamide level zero
24	17	26	10	Fusiform bacilli and spirilla	Normal	0	Normal	Massive involvement of both tonsils; right healed in 7 days; poor oral hygiene; involvement of gum; recurrence on left side in 1 week; blood sulfanilamide level zero
25	4	3	4	Fusiform bacilli and spirilla	Str. haemolyticus	.....	.....	
26	10	12	7	Fusiform bacilli and spirilla	Str. haemolyticus	0	Str. haemolyticus	Blood sulfanilamide level zero
27	7	9	3	Fusiform bacilli and spirilla	.....	.....	.....	Blood sulfanilamide level zero
28	9	9	3	Fusiform bacilli and spirilla	Str. viridans, Staph. aureus	.....	.....	
Average.....	7.0	8.9	4.7					

TABLE 4.—*Control Patients with Vincent's Ulcerative Tonsillitis*

Patient	Days of Treatment	Days Before Cessation of Pain	Bacteriologic Findings in				Comment
			Smear Before Treatment	Culture Before Treatment	Smear After Treatment	Culture After Treatment	
1	5	4	Fusiform bacilli and spirilla	Normal	0	Normal	
2	5	4	Fusiform bacilli and spirilla	Normal	0	Normal	
3	8	6	Fusiform bacilli and spirilla	Str. haemolyticus	Fusiform bacilli and spirilla	Str. viridans	
4	5	4	Fusiform bacilli and spirilla	Str. haemolyticus, Str. viridans	Fusiform bacilli	Str. viridans	
5	15	11	Fusiform bacilli and spirilla	Normal	0	Normal	Epiglottic ulcer and involvement of gum
6	16	8	Fusiform bacilli and spirilla	Str. haemolyticus, pneumococcus	.....	.....	Massive unilateral involvement
7	16	7	Fusiform bacilli and spirilla	Neisseria	.....	.....	Massive unilateral involvement
8	9	7	Fusiform bacilli and spirilla	Str. viridans	.....	.....	
9	8	7	Fusiform bacilli and spirilla	.....	.....	.....	
10	8	7	Fusiform bacilli and spirilla	.....	.....	.....	
11	9	7	Fusiform bacilli and spirilla	.....	.....	.....	
12	5	4	Fusiform bacilli and spirilla	.....	.....	.....	
13	11	9	0	Str. haemolyticus	.....	.....	
14	13	9	Spirilla	.....	.....	.....	Involvement of gum
15	5	4	Fusiform bacilli and spirilla	.....	.....	.....	
16	7	6	Fusiform bacilli and spirilla	Str. viridans	.....	.....	Involvement of gum; oxophenarsine hydrochloride, 1 injection
17	11	7	Fusiform bacilli and spirilla	.....	.....	.....	Complicated by mumps; oxophenarsine hydrochloride, 3 injections
18	8	7	.....	.....	.....	.....	Oxophenarsine hydrochloride, 1 injection
19	8	7	.....	.....	.....	.....	Oxophenarsine hydrochloride, 1 injection
20	12	9	Fusiform bacilli and spirilla	.....	.....	.....	Oxophenarsine hydrochloride, injection
21	5	4	Fusiform bacilli and spirilla	.....	0	.....	Oxophenarsine hydrochloride, 1 injection
22	7	6	.....	.....	.....	.....	Oxophenarsine hydrochloride, 1 injection
23	5	4	0	Normal	0	Normal	Oxophenarsine hydrochloride, 1 injection
Average	8.7	6.4					

Most of the patients for whom bacteriologic cultures and smears were not made were patients treated in the outpatient clinic.

the therapy was as follows: (1) All 23 patients received a dusting of the lesions with sodium perborate powder and a half-strength hydrogen peroxide gargle alternately every hour, and (2) 8 patients received, in addition to the sodium perborate and hydrogen peroxide regimen, at least one single intravenous injection of 0.06 Gm. of oxophenarsine hydrochloride. The average length of time of treatment of the control group treated with oxophenarsine hydrochloride was 7.9, compared with 9.2 days required for the remaining control group. It is believed, however, that the patients treated with oxophenarsine hydrochloride were too few to permit inferences to be drawn from these figures.

No evidence of sulfanilamide was found in the blood of 8 patients whose blood was examined for the drug. Blood was taken for this purpose two hours after administration of the powder.

There was evidence of coincident gingivitis due to Vincent's organisms in 7 patients of the entire series.

The bacteriologic studies of these groups of patients disclosed the following findings: Among the combined groups, numbering 51 patients, there were only 2 patients whose bacteriologic smears failed to reveal the presence of the well known combination of fusiform bacilli and Vincent's spirilla. Cultures of material from the throats of 15 of the 22 sulfanilamide-treated patients for whom such cultures were made showed in addition pathogenic micro-organisms. The same criteria for pathogenicity were used in these instances as in the cases of acute tonsillitis (tables 3 and 4).

ACUTE PHARYNGITIS

Nine patients with acute pharyngitis, uncomplicated by involvement elsewhere, were treated by insufflation of sulfanilamide powder. These patients presented the usual picture of sore throat, dysphagia, temperature rising to 103 F. and a hyperemic pharynx with swollen follicles on the posterior wall. The excellent results obtained in these patients were unexpected. In 7 of them a clinical cure was obtained within twenty-four to thirty-six hours after one or two insufflations of the powder. In the remaining 2 patients a similar satisfactory result was obtained at the end of seventy-two and ninety-six hours after four and six insufflations of the powder, respectively. It should be noted that the powder remained on the posterior pharyngeal wall of these patients for a short period.

Although this series is indeed too small from which to draw conclusions, our experience with insufflations of sulfanilamide in this group justifies future trial of such insufflations in the treatment of acute pharyngitis.

COMMENT

The use of sulfanilamide powder in the throat in cases of acute pharyngeal infection has received favorable comment in the literature.

Our observations show that the local application of sulfanilamide powder, insufflated either by an air-compressed or a hand-compressed pump, is effective in the treatment of patients with acute tonsillitis, Vincent's ulcerative tonsillitis or acute pharyngitis. The results achieved were not startling but, according to our statistics, were better than those observed with the standard methods of treatment. The average length of time which the patient spent in the hospital was less among those treated by insufflations of sulfanilamide powder. An interesting observation was that pain and dysphagia disappeared much sooner among the patients treated by this method.

Of military significance is the simplicity of administration and the ease of application which this form of therapy offers. It is for this reason that the present report is being made even though the number of patients treated do not permit definitive conclusions. From our experiences with this method we believe that the treatment of patients with acute tonsillitis, Vincent's ulcerative tonsillitis or acute pharyngitis by insufflations of sulfanilamide powder should be encouraged.

The blood of 16 of the patients who received the drug by insufflation was tested for the presence of sulfanilamide to determine whether any significant absorption of the drug occurred. No detectable amount of sulfanilamide could be found in the blood stream. Thus, it seems unlikely that untoward systemic complications could result from this use of the drug unless the patient was unusually sensitive to sulfanilamide. There was no evidence of sensitivity to the drug in any of our patients. It was roughly computed that about 5 grains (325 mg.) of sulfanilamide powder was administered with each treatment.

The observations on Vincent's ulcerative tonsillitis suggest interesting bacteriologic considerations. As has been observed by others, a high percentage of the patients with this disease showed association of the fusiform bacilli and Vincent's spirilla with pyogenic micro-organisms. In 15 of 22 whose ulcers were studied bacteriologically, a pathogenic micro-organism was found to be present in addition to the fusiform bacilli and Vincent's spirilla. It may be significant that a number of our patients complained of soreness in the region of the tonsil several days before the ulceration appeared. Thus, once more the possibility is suggested that tissue resistance may be lowered by pyogenic micro-organisms and that the growth and pathogenicity of the fusiform bacilli and Vincent's spirilla depend on the additional presence of pyogenic micro-organisms. The effectiveness of sulfanilamide powder in the treatment of Vincent's ulcerations of the tonsil tends to support this hypothesis.

The question may be raised whether the therapeutic effect of the sulfanilamide powder was due to local response or was the result of ingestion and systemic absorption of the powder, even though the amount

was small. About 5 grains (325 mg.) was insufflated with each treatment, which means that patients in the latter half of the series received 10 grains (650 mg.) of sulfanilamide a day. This is a question which will have to be solved by future work along these lines. It might be that sulfanilamide administered by insufflation has the combined advantage of local and, ultimately, systemic action. We are inclined to believe that the local action is the more significant.

SUMMARY

Insufflations of sulfanilamide powder were used in the treatment of 69 patients with acute pharyngeal infections. Early in the series the powder was insufflated once a day and in the latter part of the series twice a day. At present, whenever possible, the powder is administered twice a day. Fifty-four patients treated by other currently accepted standard methods were used as controls.

The average length of time required for clinical cure to be obtained was 1.8 days shorter in the series of patients with acute tonsillitis who were treated with sulfanilamide powder than in the control series, and 1.7 days shorter in the series of patients with Vincent's ulcerative tonsillitis who were similarly treated than in the control series.

Sulfanilamide insufflations appeared effective in a small group of 9 patients with acute pharyngitis, but the number of patients was too small to permit any comment.

The method of insufflation of sulfanilamide powder is simple and particularly suitable for use in the treatment of military personnel. It is for this reason especially that the present report is being made, even though the number of patients treated does not warrant definitive conclusions. We are impressed with the effectiveness of the treatment and the clinical results.

The amount of the powder administered at each insufflation was about 5 grains (325 mg.). In the cases of Vincent's ulcerative tonsillitis, the powder remained in contact with the ulcer for as long as three hours after insufflation.

The blood of 16 patients was examined for sulfanilamide; none of these patients had any detectable amount of the drug in the blood stream.

Streptococcus haemolyticus (beta) was the principal micro-organism found in acute tonsillitis. It was isolated from the tonsils of 31 of the 64 patients with acute tonsillitis.

The fusiform bacilli and Vincent's spirilla were demonstrated in smears of material from the throats of 49 of the 51 patients with ulcerative tonsillitis. Fifteen of 22 patients whose throats were studied bacteriologically were shown by cultures of material taken from the throat to harbor in addition pyogenic micro-organisms in large numbers.

XANTHOMA OR LIPOID GRANULOMA OF THE TEMPORAL BONE (HAND-CHRISTIAN-SCHÜLLER SYNDROME)

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CHICAGO

XANTHOMA of the skin and of the tendons has been known for a long time. Xanthoma of the eyelids is seen as raised yellow patches and is termed "xanthelasma palpebrarum." Similar plaques are occasionally seen in the buccal mucosa. The real nature of xanthoma was not understood until about two decades ago.

Credit is due the dermatologists who have made histologic studies of xanthoma of the skin and thus have paved the way toward the recognition of similar lesions in the internal organs, the skull and the paranasal sinuses. An extremely interesting literature on various phases of the subject has been published, but the limited scope of this paper permits only brief reference to it.

The otologist's interest in xanthomatosis lies in the fact that the temporal bone becomes affected in a great number of the cases of xanthoma of the skull. Besides the lesions in the skull, the reported cases have often presented a characteristic syndrome, such as exophthalmos, polyuria and polydipsia, and have been grouped as instances of Hand-Christian-Schüller disease. There are numerous cases of xanthoma of the skull, including the one reported here, which do not present the aforementioned syndrome. Because of the similar histologic changes, it is difficult to dissociate the two. Thus, one may refer to xanthoma or to lipoid granuloma of the skull with or without the Hand-Christian-Schüller syndrome.

THE HAND-CHRISTIAN-SCHÜLLER SYNDROME AND ITS CONNECTION WITH XANTHOMATOSIS

Between 1915 and 1930 Arthur Schüller,[1] of Vienna, Austria, described 3 cases of multiple defects of the skull, irregular in outline, with sharp demarcations, a condition which he had termed "geographic

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Read at the meeting of the Chicago Laryngological and Otological Society, April 3, 1944.

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1. Schüller, A.: (*a*) Ueber eigenartige Schädeldefekte im Jugendalter, Fortschr. a. d. Geb. d. Röntgenstrahlen **23**:12, 1915; (*b*) Ueber ein eigenartiges Syndrom von Dyspituitarismus, Wien. med. Wchnschr. **71**:510, 1921.

skull." The 3 patients were children. The first patient presented signs of dystrophia adiposogenitalis, dwarfism, exophthalmos and discharging of the ear, which had continued for a period of three years; the second, diabetes insipidus and exophthalmos; the third, exophthalmos, dystrophia adiposogenitalis and diabetes insipidus.

In 1920 Henry A. Christian [2] reported the case of a girl 5 years old in whom the outstanding symptoms were diabetes insipidus, exophthalmos and extensive defects of the skull. Thus the term Christian-Schüller disease had its inception.

It was later recognized that this symptom complex had been described by Alfred Hand,[3] who, in addition, reported the autopsy observations on his patient, a 3 year old boy. The multiple soft, yellow, discolored defects in the skull and the diseased vertebra were interpreted at that time as a tuberculous process. Because of the similarity of his case to those which had been reported by Schüller and Christian, the term "Hand-Christian-Schüller disease" has come into use. Schüller and Christian had attributed the defects of the skull to a disease of the hypophysis, while Hand doubted that the symptom complex represented dyspituitarism. None of these authors, however, connected that syndrome with xanthomatosis.

In 1922 Griffith [4] reported a case of a 9 year old patient who had multiple defects of the skull, exophthalmos and diabetes insipidus, i. e., the Hand-Christian-Schüller syndrome, as well as nodular xanthomatous lesions of the skin, the palms, the axillas and the knees. In 1924 the autopsy observations on Griffith's patient were reported by Weidman and Freeman.[5] After the calvarium was stripped, multiple defects were observed in the skull, and in one place a defect led to the brain substance. Xanthomatous changes were also found in the pituitary gland, the pineal body, the tuber cinereum, the liver and the lungs. Thus the connection of Hand-Christian-Schüller disease with xanthoma was first suggested. In 1928 Rowland,[6] on the basis of the case described in the report just cited and 2 of his own, in 1 of which an autopsy was made and the process studied by Warthin, conclusively established that the lesions of the skull in Hand-Christian-Schüller disease were xanthomatous in origin. His discussion of the etiologic

2. Christian, H. A.: Defects in Membranous Bone, Exophthalmus and Diabetes Insipidus: An Unusual Syndrome of Dyspituitarism, M. Clin. North America **3**:849 (Jan.) 1920.

3. Hand, A.: Defects of Membranous Bones, Exophthalmus and Polyuria in Childhood: Is It Dyspituitarism? Am. J. M. Sc. **162**:509 (Oct.) 1921.

4. Griffith, J. P. C.: A Case of Xanthoma Tuberosum, with Early Jaundice and Diabetes Insipidus, Arch. Pediat. **39**:297 (May) 1922.

5. Weidman, F. D., and Freeman, W.: Xanthoma Tuberosum, Arch. Dermat. & Syph. **9**:149 (Feb.) 1924.

6. Rowland, R. S.: Xanthomatosis and Reticulo-Endothelial System, Arch. Int. Med. **42**:611 (Nov.) 1928.

and pathologic aspects of this condition forms the basis of a most interesting monograph. He also maintained that Niemann-Pick disease and Gaucher's disease are not distinct entities but manifestations of xanthomatosis of the internal organs.

Sosman [7] in 1932 offered the following classification of cases of xanthoma as five clinical types:

1. Gaucher's disease—affecting chiefly spleen, occurring at any age, more frequently in female children, familial and fairly benign.
2. Niemann-Pick disease—involving the liver and the spleen, occurring in infants of the Jewish race, fatal.
3. Hand-Christian-Schüller disease—occurring in young children, affecting bones of the skull and other bones, frequently accompanied with diabetes insipidus, exophthalmos and gingivitis, less commonly with dwarfism, adiposogenital dystrophy and jaundice, not racial, fatal in one third of the cases.
4. Xanthoma of pregnancy, of icterus and of diabetes
5. Essential xanthomatosis

} not racial, occurring in adults, benign.

ETIOLOGIC AND PATHOLOGIC ASPECTS

The etiologic factors of xanthoma or lipoid granulomatosis are unknown. The lesions have often followed trauma or an acute infectious disease. Since both these conditions often occur in children, their importance can be greatly discounted. Lane and Smith [8] reported 4 fatal cases in which lesions appeared in the skull. One of the patients had a congenital type, in which lesions of the skin appeared at birth. This patient was 6 weeks old at the time of death. Chester and Kugel,[9] after reviewing 50 cases, found that 33 patients were in the first decade of life; 50 per cent of the patients were less than 5 years old; the oldest patient was 55 years of age; there were 2 males to 1 female; members of the Jewish race predominated.

The genesis of the lesions of xanthoma, according to Rowland, is due to a state of hypercholesteremia. The granuloma is a result of a hyperplastic reaction of the reticuloendothelial cells. An infiltration of lipoids, which are in excess in the body fluids, takes place in the

7. Sosman, M. C.: Xanthomatosis (Schüller-Christian Disease, Lipoid Histiocytosis), J. A. M. A. **98**:110 (Jan. 9) 1932.

8. Lane, C. W., and Smith, M. G.: Cutaneous Manifestations of Chronic Lipoidosis (Hand-Schüller-Christian Disease), Arch. Dermat. & Syph. **39**:617 (April) 1939.

9. Chester, W., and Kugel, V. H.: Lipoid Granulomatosis (Type Hand-Schüller-Christian), Arch. Path. **14**:595 (Nov.) 1932.

manner of phagocytosis. Letterer [10] and Paul Freud and associates [11] expressed the opinion that a state of deficiency of the reticuloendothelial system is the factor in the pathogenesis. They maintained that reticuloendothelial proliferation is the primary cause, that lipoidosis is a secondary manifestation and that it is the intracellular disturbance of lipoid metabolism within the endothelial cell and not phagocytosis which is responsible for the appearance of cholesterol within the proliferating cells. Freund and Ripps [12] described the case of a child 7 weeks old with generalized lymphadenopathy due to reticuloendotheliosis. Seven months after the onset of the disease, defects developed in the skull. After death, which occurred at the age of 2 years, typical lipoid granulomatosis was found in the removed glands. This convinced them that the cholesterol which was deposited within the granuloma was not responsible for its formation.

Merritt and Paige [13] reported a case in which there was severe cutaneous involvement with few cells containing fat or lipoids. At necropsy, the spleen and the lymph glands showed extensive hyperplasia of the reticuloendothelial cells without evidence of lipoid storage, while the dura, the lungs, the bones and the thymus presented typical xanthomatous lesions.

In support of the theory that the cholesterol is not precipitated from the excess lipoids of the blood is the proof that in most cases a high level of cholesterol in the blood stream is not shown. Also, the postmortem examination of organs of patients with xanthoma does not show an excessive amount of total fat in them.

Grossly the tissue is soft, yellow or yellow-brown, and microscopically the cytoplasm of the reticuloendothelial cells has a foamy appearance. There are cellular elements of chronic inflammation in various stages of fibrosis and giant cells. The latter often surround areas of cholesterol crystal formation. This constitutes the lipoid granuloma. The endothelial cells, in the process of preparation of the specimen, lose the fat globules, and what remains of them gives the cells a foam-like appearance. The term "xanthoma cells" or "foam cells" is used to describe them.

SYMPTOMS AND DIAGNOSIS

Xanthomatous lesions involving the temporal bone may exist for a long period without producing any symptoms and be discovered accidentally on roentgen examination of the skull. In some, a tumefac-

10. Letterer, E.: Allgemeine Pathologie und pathologische Anatomie der Lipoidosen, Verhandl. d. Gesellsch. f. Verdaungskr. (1938) **14**:12, 1939.

11. Freud, P.; Grossman, L., and Dragutsky, D.: Acute Idiopathic Cholesterol Granulomatosis, Am. J. Dis. Child. **62**:776 (Oct.) 1941.

12. Freund, M., and Ripps, M. L.: Hand-Schüller-Christian Disease, Am. J. Dis. Child. **61**:759 (April) 1941.

13. Merritt, K. K., and Paige, B. H.: Xanthomatosis (Schüller-Christian Syndrome), Am. J. Dis. Child. **46**:1368 (Dec.) 1933.

tion over the involved area has led to investigation and diagnosis. Symptoms arise when the granuloma invades the contiguous structures and the contents of the cranial cavity. Most of the patients present a history of an insidious onset of otorrhea, which becomes chronic and resists the usual form of treatment. Pain becomes manifest only when the periosteum or the dura becomes involved. Destruction of the mastoid cells takes place in a manner similar to that in cases in which these cells are affected by Pneumococcus type III. There is little rise in temperature, while the deafness is profound. When symptoms of dwarfism, exophthalmos, polyuria and polydipsia, with or without dystrophia adiposogenitalis, precede the aural symptoms or develop at the same time, a diagnosis of xanthoma or lipoid granuloma can be made even without histologic verification.

Some of the patients, however, present symptoms referable to the ear and the mastoid process long before the described syndrome becomes manifest. Under such circumstances diagnosis is extremely difficult. Histologic studies of the tissue removed at operation may help. If the typical xanthoma cells are not found, one must depend on later onset of some signs of the syndrome described.

In the second group it is interesting to cite the first operative case on record, which was reported by Grosh and Stifel [14] in 1923, before this entity was understood. In their patient at the age of 6 years a painless discharge of the left ear developed with infection of the left mastoid process, which was opened and drained. Seven weeks after this operation, the patient began to have marked thirst, polyuria and exophthalmos of the left eye, while a soft spot, 5 by 10 cm., was felt in the right temporal bone.

Tucker [15] recently reported that his patient, a child, had a discharging ear at 5 months of age and underwent mastoidectomy at 2 years. The wound did not heal, and thereafter polyuria developed as well as new defects in the skull. Despite the failure to demonstrate the typical xanthoma cells in the removed tissue, the clinical signs which followed were sufficient proof of the presence of lipoid granuloma.

That a therapeutic test is an aid in diagnosis is suggested by the report of Sparrow and Fetner.[16] Their patient, a child 2 years of age, who had sustained an accidental injury of the head, later presented a soft defect of the skull. The defect kept on increasing in size.

14. Grosh, L. C., and Stifel, J. L.: Defects in the Membranous Bones, Diabetes Insipidus, and Exophthalmos, with Report of a Case, Arch. Int. Med. **31**:76 (Jan.) 1923.

15. Tucker, J.: Hand-Schüller-Christian Disease (Idiopathic Xanthomatosis), Cleveland Clin. Quart. **10**:55 (July) 1943.

16. Sparrow, T. D., and Fetner, L. M.: Xanthomatosis, J. A. M. A. **99**:2176 (Dec. 24) 1932.

There were no other defects of bones, nor any general symptoms such as polyuria and thirst. Because of the disappearance of the defect following roentgen treatment, the authors maintained that the case was one of xanthoma.

In adults the symptoms may simulate those of cancer, that is, headache, irritation of the labyrinth and facial paralysis—with or without otorrhea.

Studies of the blood are not diagnostic. Lighty [17] reported a case of xanthoma with aplastic anemia, a hemoglobin value of 15 per cent and a red blood cell count of 900,000; most of the authors have reported normal blood findings, and the cholesterol content of the blood has in most instances been reported within normal limits.

Roentgenograms showing single or multiple lesions with peculiar scalloped (mouse-eaten) borders or geographic patterns are suggestive of xanthoma. However, single lesions which have no sharp borders or patterns have also been observed which simulate osteogenic sarcoma or myeloma.

The most conclusive diagnostic finding is that of xanthoma cells in a biopsy section or a section of tissue removed at operation. Through errors in histologic interpretation the removed tissues have been reported by Sosman,[7] Davison [18] and Grady and Stewart [19] as showing carcinoma, neoplasm of the reticuloendothelial system and sarcoma, respectively. As recently as 1940, a diagnosis of carcinoma simplex was made in a case reported by Rosenwasser.[20] Because of the peculiar, rapid recovery following roentgen therapy, a restudy of the microscopic sections was made. The case proved to be one of lipoid granuloma.

PROGNOSIS AND TREATMENT

Sosman [7] reported spontaneous cure without treatment in the original case of Christian. Recurrence of the lesion has been reported after apparent arrest following roentgen therapy. In other instances, new lesions have appeared after healing of the original lesion. The prognosis of life in the patient with solitary xanthoma of the temporal bone is good. In some who have been subjected to a simple or a radical mastoidectomy or to reoperation the lesions have healed, while in others there were persistent fistulous tracts. In children, xanthoma of the skull

17. Lighty, D. E.: Lipoid and Lipoid Diseases: Xanthomatosis, Arch. Int. Med. **53**:379 (March) 1934.

18. Davison, C.: Xanthomatosis and the Central Nervous System (Schüller-Christian Syndrome), Arch. Neurol. & Psychiat. **30**:75 (July) 1933.

19. Grady, H. G., and Stewart, H. L.: Hand-Christian-Schüller Disease and Tuberculosis, Arch. Path. **18**:699 (Nov.) 1934.

20. Rosenwasser, H.: Lipoid Granulomatosis (Hand-Schüller-Christian Disease) Involving Middle Ear and Temporal Bone, Arch. Otolaryng. **32**:1045 (Dec.) 1940.

usually appears in multiple foci, and when the Hand-Christian-Schüller syndrome is presented, the mortality is about 35 per cent. The prognosis is still graver when the lungs, the liver or the brain becomes involved. Surgical treatment of the lesion hastened the downhill course in some of the reported cases.

The treatment is limited to moderate doses of roentgen radiation, which produces fibrosis of the lesion and results in relief of symptoms. It cannot be considered, however, as affecting the basic cause of the lipoid granuloma.

Fig. 1.—Roentgenograms of the right and left mastoid processes, showing an area of destruction of the left external auditory canal and the anterior contour of the petrous bone.

REPORT OF A CASE

C. W., Jewish, aged 36, a teacher, consulted me on May 17, 1942, because of headaches, extreme weakness, giddiness, left-sided tinnitus and deafness. He stated that the symptoms, in the order named, had come on five months previously and had gradually become more severe until it was difficult for him to do a full day's work. The headaches, which were worse at night, originated at the left temporoparietal region and radiated toward the top of the head and to the face. The attacks of giddiness usually followed mental strain; they were not produced by sudden movement or bending of the head. At no time during that period or in recent

years had he experienced earache, discharging of the ear, twitching of the muscles of the face, difficult or painful movements of the jaw, fever, loss of weight or enlargement of glands of the neck.

The important item in the past history was that he had been confined in a sanatorium about nine years previously because of pulmonary tuberculosis. He was there one year and was discharged with complete arrest of the lesion.

A recent physical examination had disclosed fibrosis of the upper lobe of the right lung. Except for a persistent increase in the number of red blood cells (6,000,000 to 6,500,000), all the findings were normal. The blood cholesterol was normal.

The examination of the left ear disclosed redness and bulging of the superior portion of the tympanic membrane and of the anterosuperior wall of the auditory

Fig. 2.—Roentgenogram taken ten weeks later, showing an extension of the process and involvement of the anterior part of the auditory canal down to the glenoid capsule of the temporomandibular joint.

canal, causing a narrowing of the lumen anteriorly. The remainder of the tympanic membrane appeared normal. There was no discharge from the auditory canal and none on aspiration with the Siegel otoscope. The right ear drum appeared normal, and the hearing was normal. The hearing on the left side was reduced to 10 feet (3 meters) for spoken voice; Weber's test showed sound lateralized to the left; bone conduction was increased. On deep pressure, an area of tenderness was elicited over the attic and the temporomandibular joint, but none over the mastoid process. A caloric test produced equal responses in the two ears. Inflation of the eustachian

tube, especially with amphetamine vapor, produced a temporary improvement in hearing. All the tests, therefore, pointed to a lesion of the middle ear.

Roentgenographic examination of the mastoid processes showed uniform moderate sclerosis of both. However, at the left squama, just above the temporomandibular joint, there was an area of rarefaction about 20 mm. in diameter with indistinct margins. No other defects of the skull were observed (fig. 1).

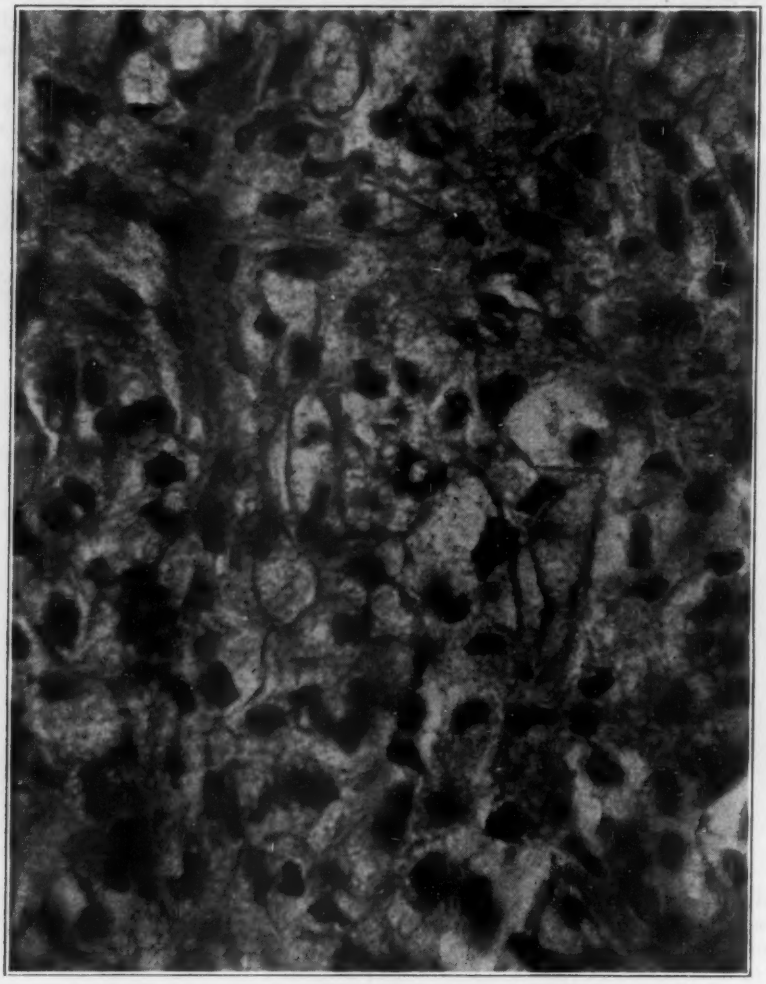

Fig. 3.—High magnification of the bone marrow, showing large endothelial cells with distinct cell borders and relatively small vesicular nuclei and foamy cytoplasm. Multinuclear giant cells are also observed.

At that time I considered the following conditions:

First, localized zygomatic mastoiditis as a result of Pneumococcus type III infection of the middle ear. However, the long afebrile period without signs of trismus or of developing intracranial complication could not be reconciled with such a lesion.

Second, tuberculosis of the skull, because of the history of pulmonary tuberculosis, The usual tuberculous infection of the middle ear, whether the bacilli have reached the ear by way of the eustachian tube or by way of the blood stream, would have, by that time, produced a discharging ear. However, tuberculous involvement could not definitely be ruled out.

Third, cancer, primary or metastatic. The absence of a loss of weight, and of involvement of the regional lymph glands and the relief of the pain with administration of a mild sedative were the contradicting factors.

Fig. 4.—Operative area. It shows a ringlike formation of new bone, following roentgen therapy.

The patient was advised by me to permit surgical exploration of that area. He returned ten weeks later. On reexamination the findings were essentially the same. Roentgenologic studies, on Aug. 10, 1942, showed an increase in the area of bone absorbed, as will be noted by comparing the films taken ten weeks apart (figs. 1 and 2).

Consideration was given to other lesions that might have their primary origin in the squama and progress toward the middle ear and the temporomandibular joint.

A preoperative diagnosis of xanthoma of the temporal bone was included with that of tuberculosis or cancer.

On August 22 I exposed the involved area by means of an endaural incision with the patient under general anesthesia. A greenish brown sequestrum was removed and with it the underlying necrotic dura, which exposed the brain substance. Further removal of necrotic bone led above to chalklike sclerosed bone, below to exposure of the capsule of the temporomandibular joint and posteriorly to the attic of the tympanic cavity. The granulation tissue which extended into the middle ear was not disturbed. Postoperatively there was only 1 degree elevation of the temperature (F.), which lasted for twenty-four hours. The area healed without suppuration.

The histologic studies of the removed sections of bone showed the pathologic process to be lipoid granuloma (xanthoma) (fig. 3). Three courses of roentgen therapy were given to the involved area, and each time a loss of hair resulted around the left temple, which subsequently recovered.

It is now two and one-half years since the onset of the patient's symptoms and twenty months since the operative procedure. Roentgenograms of the operative area taken at frequent intervals show progressive fibrosis and calcification of the defect (fig. 4). The attacks of giddiness have disappeared, but the hearing has not improved. The patient has gained weight and is working eight hours daily.

SUMMARY AND CONCLUSION

A new type of granuloma, that is, xanthoma or lipoid granuloma, must be included among those which are known to affect the temporal bone and the structures of the middle ear.

The genesis of this lesion is ascribed by some to a disturbance of fat metabolism which produces cholesterol infiltration of the reticuloendothelial cells. Others attribute it to a disturbance of the equilibrium of the fat content within the endothelial cells. The characteristic histologic feature of this granuloma is the presence of the so-called xanthoma cells or foam cells.

This type of granuloma may invade, in solitary or multiple areas, any portion of the skull, including the paranasal sinuses. If it invades the temporal bone, it may involve the squama, the petrosa and the dura, before it progresses to the air cells of the mastoid process.

The symptoms are not typical of an ordinary surgical type of mastoiditis or a cancer, which it simulates; nevertheless, clinically the differentiation is not easily made. In the presence of the systemic disturbances known as the Hand-Christian-Schüller syndrome, such as exophthalmos, diabetes insipidus and of other osseous lesions in the skull which show an irregular outline with sharp borders on the roentgen film, the diagnosis of xanthoma should not be difficult. In the presence of a solitary lesion with indefinite borders, a diagnosis can be made only on the finding of xanthoma cells in the biopsy specimen or in the tissue removed at operation. The blood count and the chemical composition of the blood are usually not changed.

It is important to keep the possibility of this lesion in mind in operative cases in which the wound does not heal well and in which other factors have been ruled out. Routine histologic studies of the tissue removed at operation may discover some cases in which the lesion heals spontaneously and others in which it runs a protracted course.

These patients should not be subjected to any surgical procedures except biopsy. Roentgen therapy has a favorable influence on the symptoms by producing fibrosis of the xanthoma.

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SOME INTERESTING LESIONS OF THE TONGUE

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EMBRYOLOGICALLY, the tongue is derived from two main sources. The anterior portion, which forms the anterior two thirds of the tongue, is derived from tissues of the first branchial region. These tissues, growing from each side of the tuberculum impar, finally enclose it. This distal portion of the tongue, formed by fusion of the lateral masses, is in reality two separate organs, each with its own blood and nerve supply, joined together in the midline by a fibrous partition or septum.

The posterior portion of the tongue is formed from tissue masses at the medial ends of the second arches. These masses fuse to form a single mass and do not have the septum common to the anterior portion.

The blood supply of the tongue is derived from the lingual artery and its branches.

The anterior portion of the tongue receives its nerve supply for taste from the chorda tympani nerve, while the lingual nerve is its nerve for ordinary sensations. The posterior third is supplied by the glossopharyngeal nerve for both taste and ordinary sensations.

The hypoglossal is the nerve of motion for the entire tongue.

One interesting fact, often overlooked, is that a portion of the posterior third is supplied by a branch from the superior laryngeal nerve. It is because of this that forceful depression of the tongue with the tongue blade too far back often causes gagging. Rhythmic traction on the tongue has been used to control hiccup through the same mechanism.

The tongue is abundantly supplied with lymphatics, which anastomose freely; thus the fact that cancer of one side may be followed by enlargement of lymph nodes on the opposite side is accounted for.

Observation and study of the tongue were once an important part of every general physical examination. The old-time physician could describe in detail the strawberry tongue of scarlet fever, the shriveled tongue of dysentery and the brown, dry tongue of typhoid fever.

Newer diagnostic methods and increased laboratory facilities have supplanted to some extent such detailed observation, but the evidence is still to be seen if one cares to look for it. The tongue is an easy organ to examine, and it often tells a story that clinches the diagnosis. It probably gives as many clues to systemic diseases as to local ones.

Read at the meeting of the Middle Section of the American Laryngological, Rhinological and Otological Society in Indianapolis, Jan. 17, 1945.

Nature has adapted the tongues of animals to meet their individual requirements.

In the fish, in which the tongue is not of importance, it is merely a fold in the floor of the mouth—but even in this animal the organ shows a purpose. In the muskellunge, toothlike appendages are present which enable the big fish to hold his prey while he turns and scales it. The cow's tongue is rough, due to cornification of its filiform papillae, which gives the organ a "nonskid" surface and enables the cow to grasp its grassy food better. The humming bird's tongue is tubular, while that of the lemur has a comblike fold on the under side, with which it cleans its wooly coat.

In man, local lesions of the tongue are mostly lesions of its epithelial covering. This very specialized membrane concerns itself with transmission of sensations to the brain. Changes of temperature, tactile sensations and registration of the four primary flavors, salt, sour, bitter, and sweet, are the function of this mucous membrane.

Generalized nervous disorders may be evidenced by certain misbehaviors of the tongue, as for instance the tremors seen in hyperthyroidism, the clumsiness of the tongue in myxedema and its purposeless movements in chorea. The paretic may protrude his tongue and forget to pull it back.

The frightened, apprehensive patient opens his mouth widely and protrudes his tongue with practiced ease when examined.

CONDITIONS OF THE TONGUE AND THEIR SIGNIFICANCE

For convenience, pathologic conditions of the tongue may be roughly classified as follows: (1) metabolic diseases; (2) infections; (3) neoplasms, benign and malignant; (4) congenital abnormalities; (5) lesions due to mechanical causes.

At times a lesion of the tongue may result from a combination of factors. For example, the leukoplakic tongue may show an area of cancerous degeneration; glossodynia may result from a variety of causes, infectious, metabolic or mechanical.

Anemia (fig. 1 *A*).—This condition is indicated by a pale tongue, since the color of the blood shines through the capillaries, which lie close to the surface. In cases of shock and extremely low blood pressure the indentations made by the teeth may remain after the tongue is protruded. A slick tongue usually means vitamin deficiency.

Coated Tongue (fig. 1 *B*).—Coating of the tongue often has little to do with the state of the digestive system. It may be a local condition due to lack of oral cleanliness. It is seen principally during severe illness or when there is some local factor which interferes with the intake of fluid and the ingestion of food. The coating represents a collection

of bacteria, epithelial debris and food particles, lodged in the interstices of the papillae, which has not been removed by mechanical means. The condition may be indicative of some general illness but is only indirectly the result of it.

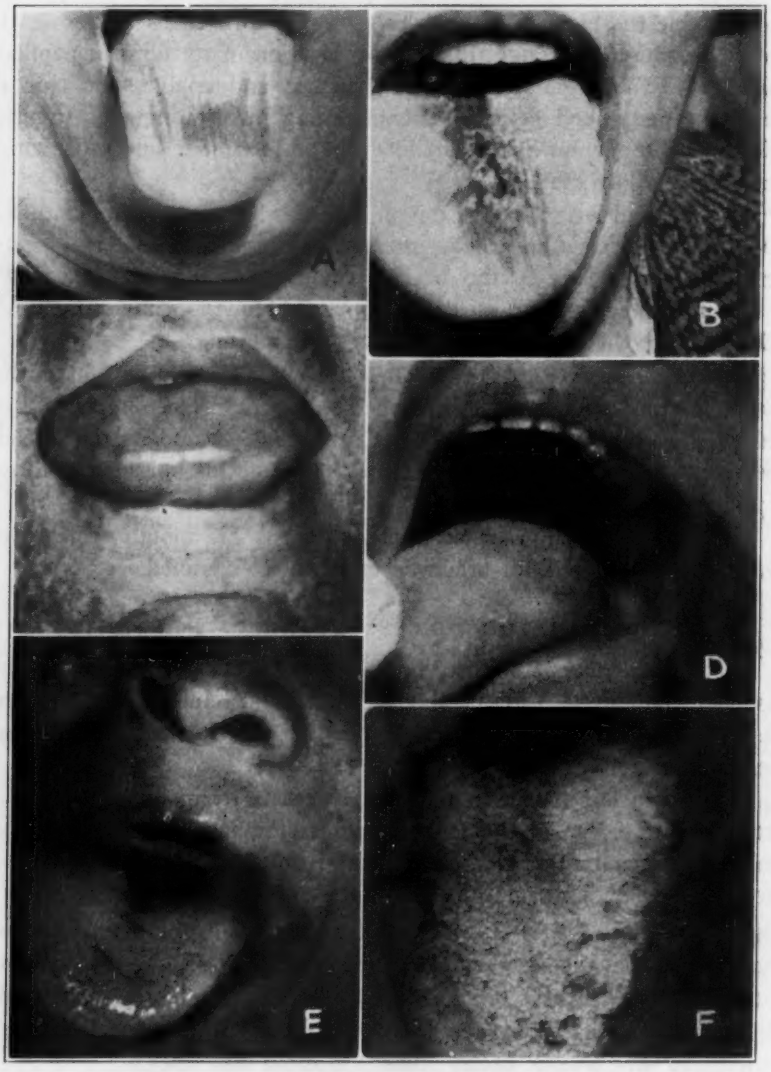


Fig. 1.—*A*, tongue of a patient with anemia; *B*, coated tongue; *C*, angioneurotic edema; *D*, irritation of the folium linguae (one of the causes of glossodynia); *E*, riboflavin deficiency; *F*, nicotinic acid deficiency—early pellagra.

Edematous Tongue (fig. 1 *C*).—Edema of the tongue is the result of infection or of allergy. It occurs in some people after they have

ingested certain foods, such as fish, walnuts or chocolate. It may result from bites and stings of insects or other injury.

The swelling is often sudden and alarming but seldom extends beyond the confines of the tongue and therefore does not threaten respiration.

The allergic type may be promptly relieved by administration of epinephrine. Cracked ice held in the mouth is helpful. Occasionally it is necessary to make an incision in the midline to relieve the congestion.

Inflammation of Folium Linguae (fig. 1 *D*).—The folium linguae is a small area on the side of the tongue just in front of the anterior pillar. Irritation of this area may be caused by a jagged tooth or by infection of the taste buds in the area. In the rabbit these taste buds are highly developed and play an important part in the animal's sense of taste. Treatment consists in removal of sources of irritation and application of a weak solution of iodine to the part. Irritation of this area is one of the causes of glossodynia.

Burning Tongue.—Glossodynia, or burning of the tongue, may be present in a normal-appearing tongue. There are several causes of this distressing condition: It is associated with various types of anemia. It is present in some vitamin deficiencies. An ill fitting or improperly vulcanized denture may cause it. It is experienced by those who smoke to excess. Malocclusion of the temporomandibular joint is a common cause of the condition. Erythema migrans (geographic tongue) may be causative. Certain drugs when used over a long period occasionally produce it. Glossodynia is present in lingual tonsillitis. Some writers believe that fillings of dissimilar metals may set up a minute galvanic current which would cause burning tongue. An interesting thing about the disease is that it bothers the patient during the day but does not interfere with his sleep. Food may or may not cause pain. Burning tongue may be associated with generalized oral burning or with alterations in taste.

Vitamin Deficiencies (fig. 1 *E* and *F*).—It is now known that many so-called dyspeptic tongues of the older writings in reality represented vitamin deficiencies.

Two of the principal deficiencies are lack of riboflavin and lack of nicotinic acid (the cause of pellagra).

Deficiency of riboflavin is possibly the commonest single vitamin deficiency, especially in children. With this condition the tongue is magenta or reddish purple and is rough and granular, the so-called "pebbled tongue." It is stated that early in the disease the tongue is coated and that later there is patchy shedding of the "fur" usually on the outer and upper surfaces. There is associated fissuring at the angles of the mouth (fig. 1 *E*), and sometimes keratitis. This condition responds readily to large doses of riboflavin.

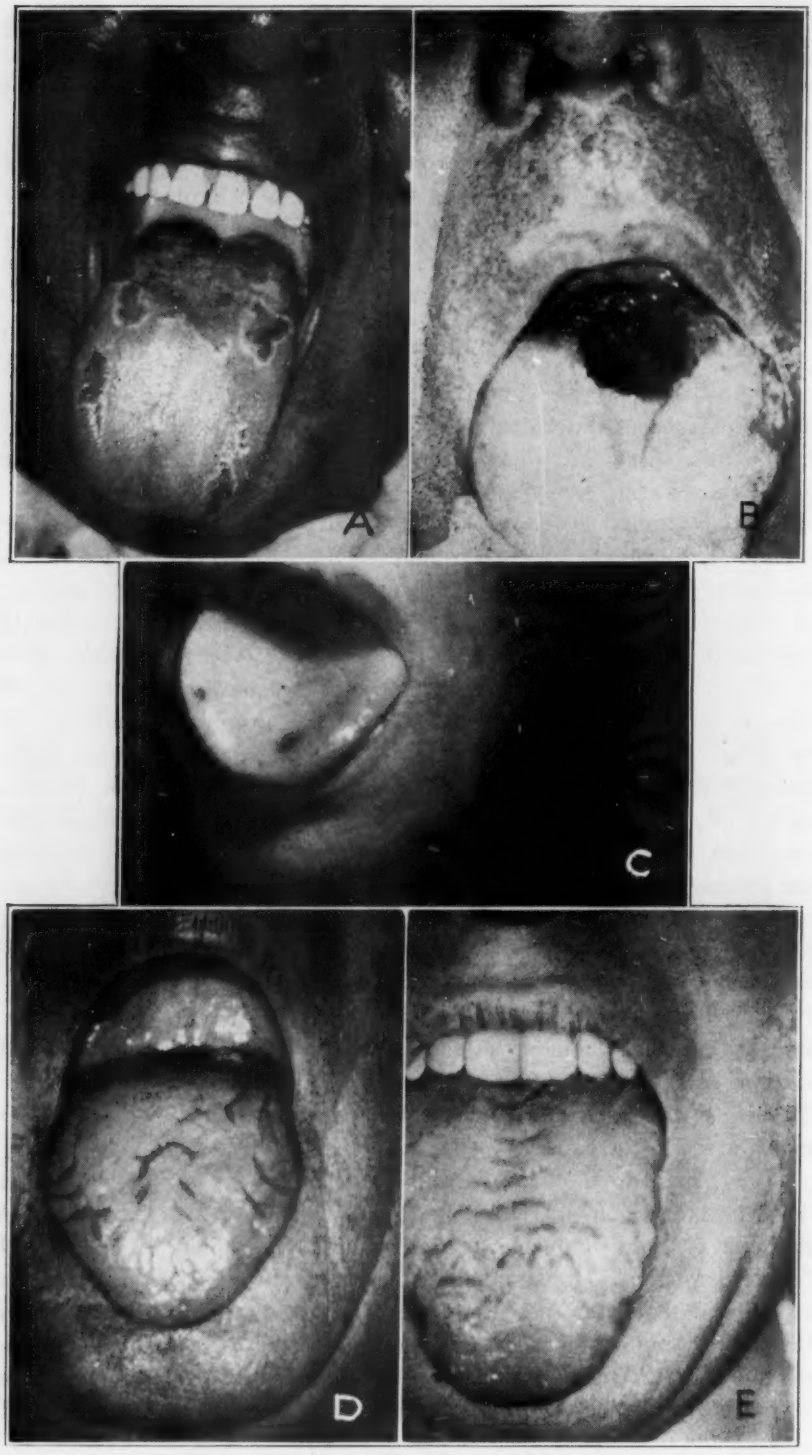


Fig. 2.—*A*, geographic tongue; *B*, leukoplakia (from which an area of cancer has just been removed); *C*, purpura; *D* and *E*, furrowed tongues of father and son.

In pellagra there are early swelling and redness of the tip and the margin of the tongue (fig. 1 *F*). Later the tongue becomes fiery red, and the papillae become enlarged. Atrophy may follow. Ulcerations sometimes appear, which may be covered with a grayish membrane containing Vincent's organisms. In some cases, however, the only objective finding is a bald, slick tongue. Following the use of nicotinic acid, improvement is rapid.

Frequently both pellagra and deficiency of riboflavin are present together.

Whenever examination reveals a red, sore tongue, it is probably best to give vitamin B complex rather than one fraction of it, as vitamin B deficiencies are often multiple.

Geographic Tongue (fig. 2 *A*).—This condition is known also as erythema migrans. It is a relatively harmless condition but causes a great deal of worry in the mind of the patient. It consists of a series of bare spots on the tongue, which are surrounded by rings of whitish fur, due to swelling and elongation of the filiform papillae around the bare spot. This condition is sometimes seen in children following exanthems and may occur in adults as a chronic condition which persists for years without symptoms. It is said to be one of the causes of glossodynia. As the involved filiform papillae are shed, a bald spot results, which is slick, shiny and painless. As time goes on, the filiform papillae regenerate, and the bald spot becomes normal again, since it now is coated as is the rest of the tongue. The lesions tend to migrate over the surface of the tongue and present an ever changing picture, hence the name "wandering rash."

Leukoplakia of the Tongue (fig. 2 *B*).—This condition is characterized by thickened whitish patches occurring on the surface of the tongue. These are due to proliferation of the superficial layers of the mucous membrane and sometimes of the filiform papillae. The condition is painless and becomes known to the patient on casual observation.

The cause is not known. The condition is aggravated by the use of irritants, and cancer may supervene if proper precautions are not taken. The principal thing to remember about treatment is that all forms of irritation must be avoided. The use of alcohol, tobacco and spices is forbidden. The diet must be bland and the mouth kept scrupulously clean. Under no circumstances should silver nitrate be used. It is probable that more cancers have been caused than lesions cured by this agent. Poorly made dentures should be removed.

Some success has been reported with the administration of large doses of brewers' yeast. If achlorhydria is present, dilute hydrochloric acid should also be given. Sore spots that develop should be surgically removed and examined for cancerous change.

It was formerly thought that there was a relationship between syphilis and leukoplakia, but later writings deny this. In any case the prognosis should be guarded.

Purpura (fig. 2*C*).—The tongue is sometimes the seat of small purplish spots which evidence generalized purpura. The spots are tiny

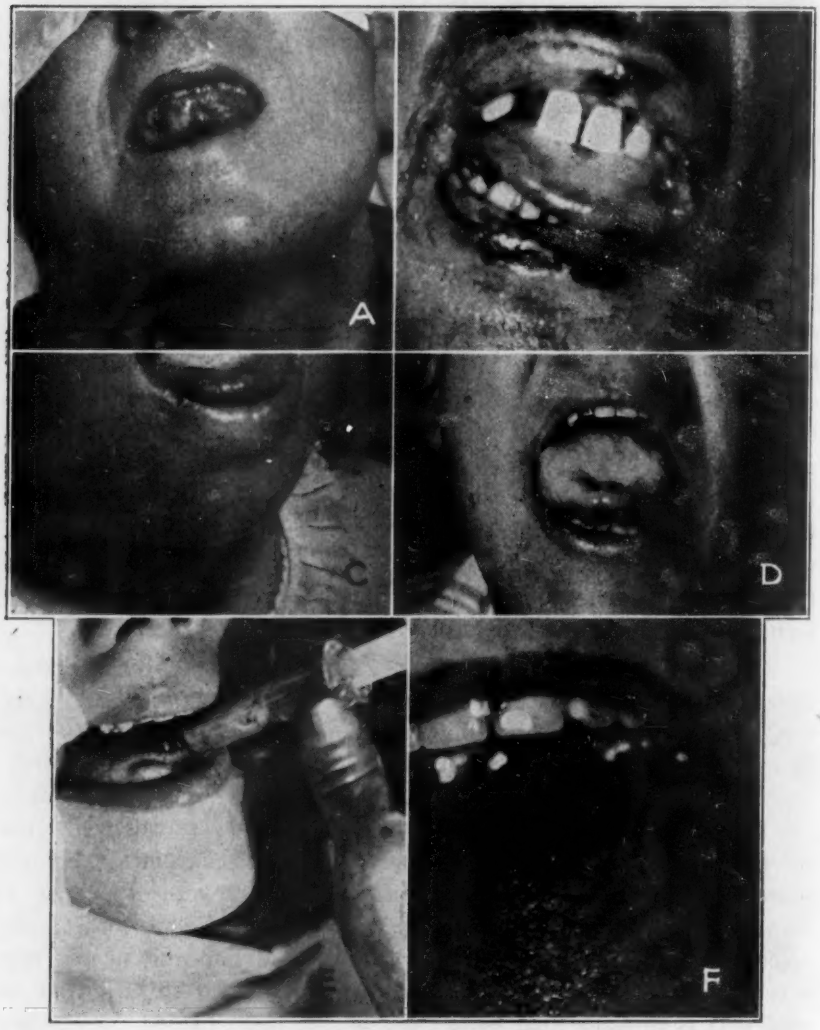


Fig. 3.—*A*, streptococcic ulcers; *B*, ulcers from dirty mouth (patient also has an abscess of the neck); *C*, canker sore; *D*, abscess of the tongue; *E*, aspiration with a large bore needle to locate an abscess of the tongue; *F*, chancre.

areas of hemorrhage resulting from rupture of capillaries in the tissues. They usually denote the presence of a serious general condition.

Furrowed Tongue (fig. 2 *D and E*).—Furrowed, grooved or scrotal tongue is a congenital familial condition of little pathologic significance. It may be seen in several members of the same family. The tongue is usually large and presents deep grooves or furrows. At times it may show mild glossitis, probably caused by food particles and debris accumulating in the cracks.

People with this condition usually have a broad dental arch with well formed teeth and seldom show achlorhydria.

Ulcers of the Tongue (fig. 3).—True ulcers of the tongue occur from infection and chronic irritation. Those due to infection (fig. 3 *A*) are best treated by cauterization and sulfonamide compounds if the organism is sensitive to the action of these drugs. Vaccines are usually ineffective. The chronic, indolent ulcers seen in the aged and the debilitated may be treated by application of 2 per cent chromic acid. It may be necessary to excise the ulcer surgically.

When ulceration occurs opposite a jagged tooth and does not heal promptly when the source of irritation has been removed, it should be surgically excised, serially sectioned and examined to make sure that cancer has not started. There is no treatment of value for the ulcer of tuberculosis. The condition seems to respond in about the same manner as the pulmonary condition which almost always accompanies it. Use of the actual cautery temporarily relieves the pain.

Canker sores (fig. 3 *C*) are small painful ulcers which may form anywhere on the tongue. There are several types. One occurs periodically in otherwise healthy adults and is probably due to an idiosyncrasy for some food, such as fish or walnuts. Until the offending food is discovered, the only treatment of value is palliative and consists of light cauterization of the lesions with Bonain's solution.[1] This sears the endings of the sensory nerves and enables the patient to eat with comfort.

A second type of canker sore occurs in young women at the menstrual period. Treatment consists in the intradermal injection of smallpox vaccine. It is probable that this type is due to a virus.

The third type is a simple ulcer which starts as a deep nodule, comes to the surface and breaks down; it occurs only once.

Abscess of the tongue (fig. 3 *D*) may result from wounds, especially puncture wounds, or infections deep in the tissues. Owing to the fact that the tongue is divided by its tough fibrous partition, an abscess seldom occurs exactly in the midline. As the abscess enlarges, pain and swelling increase, until the patient has great difficulty in swallowing. After aspiration with a large bore needle to locate the lesion (fig. 3 *E*), if it is a deep one, the abscess should be opened. Recovery is then rapid.

1. Bonain's solution consists of equal parts of cocaine crystals, phenol crystals and menthol crystals.

Chancre (fig. 3 *F*).—Chancre may occur anywhere on the tongue. It is painless and persistent, and may resemble a small punched-out ulcer or a papule. After days or weeks it is followed by mucous patches (fig. 4 *A*), and the typical rash breaks out on the chest and the flexor surfaces of the arms.

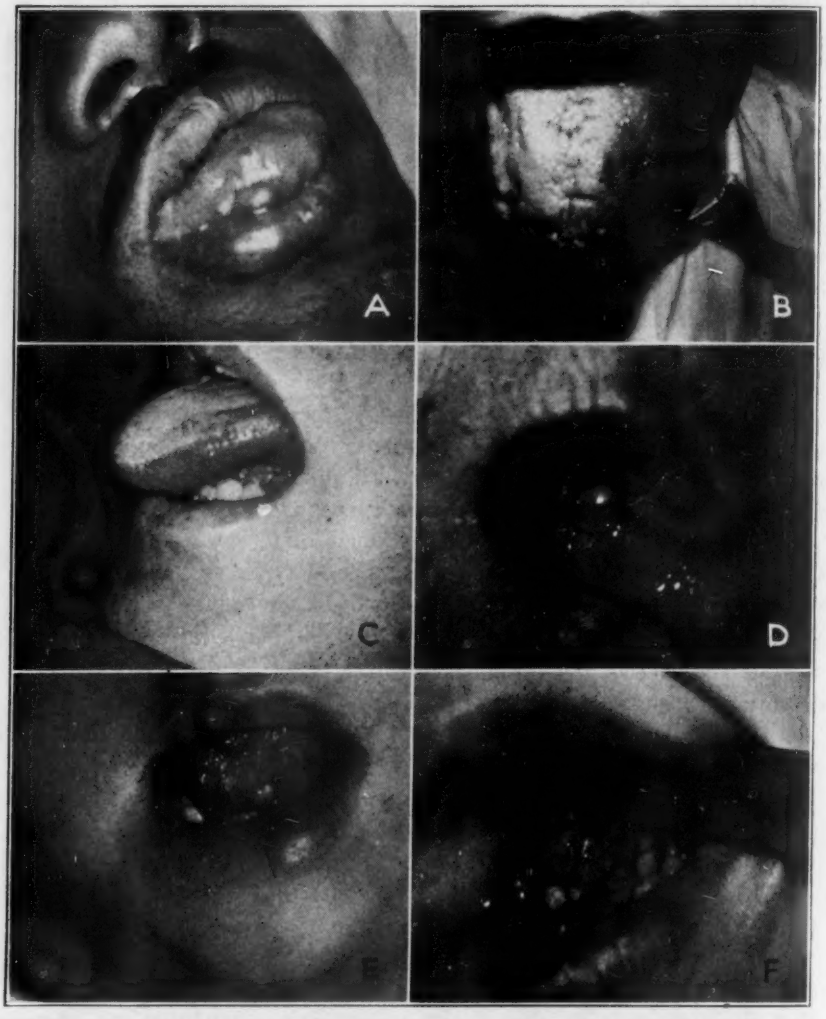


Fig. 4.—*A*, mucous patches; *B*, lesion of tertiary syphilis; *C*, papilloma; *D*, keloid; *E*, fibroma; *F*, carcinoma.

Mucous Patches (fig. 4 *A*).—These are due to syphilitic spirochetal invasion of the tiny vessels which supply the papillae. This causes the papillae to swell and become taller and enables them to hold more fur or coating. They then resemble islands of coating on the tongue. These

islands swarm with spirochetes and are highly infectious. They are usually painless.

Gumma of the Tongue (fig. 4 *B*).—The tertiary lesion of syphilis, or gumma, often occurs in the midline owing to the predilection of the spirochetes for fibrous tissue. The organisms invade the deeper vessels and set up an infiltration around them. A ball-like necrotic mass forms and slowly migrates toward the surface, where it breaks down to form a sloughing ulcer.

Papilloma (fig. 4 *C*).—Papilloma is the commonest simple tumor of the tongue. It may be met with at any age but is more frequent as age advances. It is confined to the papillae-bearing area of the tongue. In appearance it resembles a wart. It may be the result of any source of irritation. The papilloma which occurs in childhood may be snipped off and does not recur. When it occurs in later life, in association with leukoplakia, it should be regarded with suspicion, carefully removed and examined for cancer.

Keloid (fig. 4 *D*).—Keloid of the tongue is rare. Keloid is much more common in the skin and when present in the tongue it is usually in a patient who has keloids of the skin. It normally follows an injury but may arise spontaneously. Removal is generally followed by recurrence.

Fibroma (fig. 4 *E*).—Fibroma of the tongue generally occurs as a firm, rounded tumor and may be present anywhere on the tongue and may occur at any age. It is composed of fibrous tissue with few blood vessels. One form is polyp-like and edematous and may be seen in the newborn. The small, firm fibroma tends to grow slowly and does not recur when removed.

Carcinoma (fig. 4 *F*).—Carcinoma of the tongue may start in several ways. Chronic glossitis, syphilitic or otherwise, may have been present for a long time before the tiny sore first appears. An ulcer from any source is benign at first but may become cancerous if it persists. As malignant changes begin, the ulcer enlarges, its edges become harder and it becomes painful. It is the pain which attracts the patient's attention. However, early in its development cancer may be symptomless, and that of the tongue is no exception. Later, pain referred to the ear is typical. In the early stage surgical removal may produce a cure; later, irradiation is indicated.

Cyst of Salivary Glands (fig. 5 *A* and *B*).—On the under side of the tongue, 1 or 2 cm. from the tip and on either side of the midline lie the glands of Blandin and Nuhn (fig. 5 *B*). They are small salivary glands from which cysts occasionally arise. Simple incision of the cyst is not sufficient; the gland must be dissected out if recurrence is to be prevented.

Pyogenic Granuloma (fig. 5 *D*).—This tumor is a mushroom-shaped growth attached to the tongue by a stalk or pedicle. The tissue of which it is composed is similar to granulation tissue, and staphylococci usually may be cultured from it. The growth starts from an infected site of

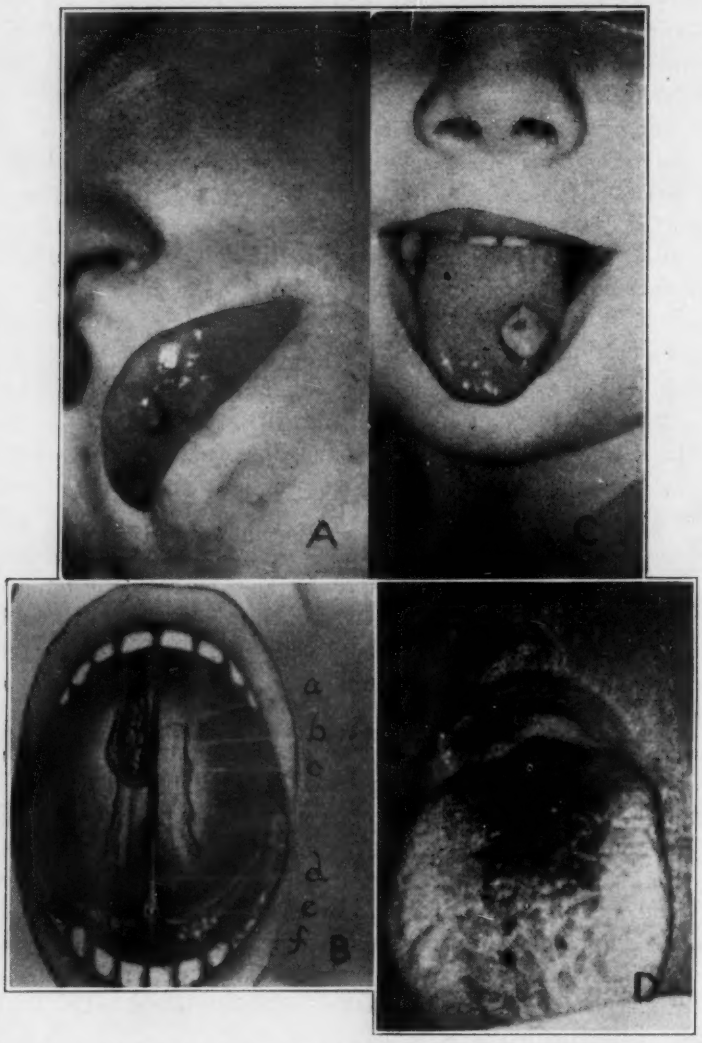


Fig 5.—*A*, cyst of gland of Blandin and Nuhn; *B*, drawing of gland of Blandin and Nuhn: *a* indicates an anterior gland of the tongue; *b*, layer of muscle cut to show the gland; *c*, plica fimbriata; *d*, frenulum linguae; *e*, duct of submaxillary gland; *f*, openings of ducts of the submaxillary gland; *C*, black or hairy tongue; *D*, pyogenic granuloma.

injury. It is grayish-pinkish white and fairly vascular. Removal must be complete, with cauterization of the base, or the condition tends to recur.

Black Tongue (fig. 5 *C*).—Hairy or black tongue is an interesting condition which occurs when the filiform papillae in the rhomboid area of the tongue become elongated and overgrown with the fungus called Aspergillus niger (fig. 5 *C*). The fungus may become as much as an inch long but causes no symptoms until it sets up an irritation and tickling in the throat. Chance observation of the condition will also bring the patient hurriedly to the doctor. Treatment consists in shaving off the growth with a dull knife and applying a 2 per cent solution of salicylic acid or a 3 per cent iodine solution. There is also a red and a yellow type of hairy tongue, with the same symptoms and treatment.

505 Hume-Mansur Building.

Case Reports

ANOMALOUS ANTRAL TOOTH

CAPTAIN PAUL E. NOONAN, MEDICAL CORPS, ARMY OF THE UNITED STATES

The anomaly observed is presented primarily for its interest as a rare embryologic defect. In addition it emphasizes again the need for close cooperation between the dental surgeon and the otolaryngologist.

REPORT OF A CASE

In September 1944 a 24 year old soldier noticed a foul taste in his mouth, which he attributed to a "wisdom tooth." About one month later he was struck rather forcefully in the face while boxing. One week after sustaining this trauma, the left side of his face became swollen and somewhat tender, and he went to the dental clinic, thinking his trouble originated in the aforementioned molar tooth. During his examination at the dental clinic, a fistula was found in the region of the left upper third molar tooth. Incision and drainage were performed at that time. Dental roentgenograms did not reveal the presence of a molar tooth in this area, although there was no history of previous extraction. The acute inflammation was treated at the dental clinic; then he was referred to the otolaryngologic clinic at the station hospital for further examination and treatment.

Physical Examination on Admission to the Hospital.—The patient was a white man, 6 feet 1½ inches (187 cm.) tall and weighing 175 pounds (79.5 Kg.). He was not acutely ill. His skin was clear. His pupils were regular, equal and reactive to light and in accommodation.

The mucosa of the left nasal fossa was moderately inflamed and edematous. The left inferior turbinate was large and boggy. There was no demonstrable discharge. Transillumination revealed a cloudy left antrum.

The mouth revealed an alveolar fistula posterior to the left upper second molar tooth, from which there was foul-smelling purulent drainage. A probe could be inserted into this opening with ease for about 2.5 cm.

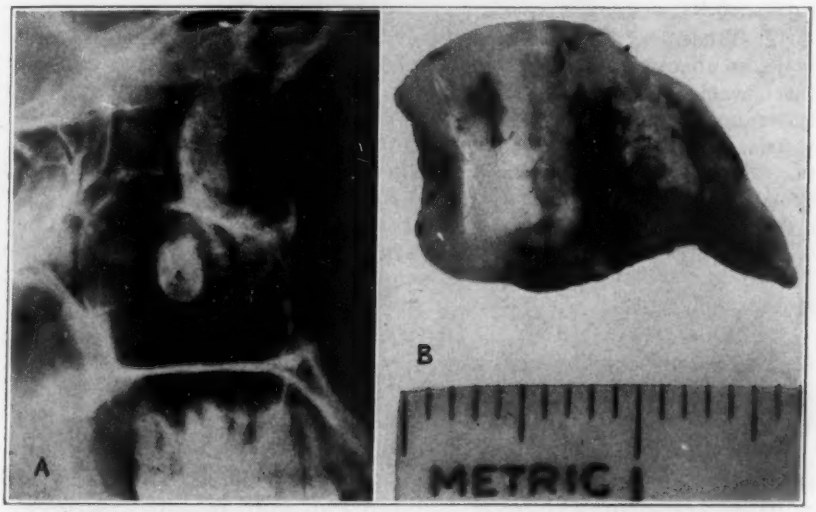
The remainder of the physical examination gave entirely negative results.

Laboratory Data.—Cultures of material taken from the washings of antral irrigation and from the fistula revealed the presence of Streptococcus viridans and Staphylococcus albus. Urinalysis gave negative results; the blood count was: red blood cells, 5,400,000; white cells, 8,650, of which 63 per cent were neutrophils 36 per cent lymphocytes and 1 per cent monocytes.

Roentgen Report.—"On posterior-anterior projection the left antrum appears to be somewhat hazy. At the medial superior angle of the left antrum is an area of increased density which has the appearance of a tooth. (See *A* in figure.) On lateral view the antral haziness described on posterior-anterior projection is not noted. However, there is an area of increased density, measuring 1 by 1.5 cm. The central portion of this structure appears dental and seems to be located in the region of the posterior and mediosuperior wall of the left antrum. The root of this toothlike structure appears to be posterior and inferior to the floor of the left orbit."

Course in Hospital.—On November 9 the left antrum was irrigated by puncture through the inferior meatus. A large amount of foul-smelling purulent discharge was in the washing. At this same time iodized poppyseed oil 40 per cent was injected into the fistula, and roentgen examination revealed that the antrum contained the radio-opaque medium, but the fistula itself was not well visualized. On the following three days the antrum was irrigated through the antroalveolar fistula with recovery of a large amount of purulent fluid.

On November 13 the Caldwell-Luc operation was performed. Anesthesia was obtained by blocking the left maxillary nerve with an injection of 1 cc. of a 2 per cent solution of procaine hydrochloride made into the left pterygomaxillary fossa by the extraoral route. The anterior wall of the antrum was removed in the usual manner. The antrum was found to be filled with a thick purulent discharge, and the mucosa was hypertrophied throughout the sinus. The protruding crown of a tooth was visible at the junction of the posterior and superior walls of the antrum. The tooth was deeply and firmly embedded and was removed by gradual



A, lateral view of the skull showing the antral location of the anomalous tooth. *B*, extracted tooth.

elevation with periosteal elevators. When extracted, it was found to be a molar tooth with a single rudimentary root but a well developed crown (*B* in figure). This tooth was surrounded by necrotic mucosa, and after it was removed, a distinct compartment was observed in which it had been lodged. All mucosa of the sinus was then curetted away. The antral opening of the fistula could not be visualized, as it was located just behind the anterior wall of the sinus. It could be definitely found by probing, however, and was thoroughly cleaned out with a curet. A large nasoantral window was then made into the inferior meatus after removing the anterior tip of the inferior turbinate, which was large and obstructive. The inferior margin was removed down to the floor of the nose. Iodoform gauze was placed in the antrum and drawn out through the nose, and the incision of the canine fossa was closed with black silk.

Following the operation, the convalescence of the patient was entirely uneventful. The antroalveolar fistula promptly closed, and all discharge from the window completely subsided.

MAXILLARY SINUSITIS AND ETHMOIDITIS OF THE RIGHT SIDE DEVELOPING DURING INTRAMUSCULAR INJECTION OF PENICILLIN FOR PANSINUSITIS OF THE LEFT SIDE

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THE case to be reported is of particular interest because of the following observations:

1. Porosity of the anterior and posterior walls of the left frontal sinus disappeared and acute pansinusitis of the left side cleared up completely except for a cyst in the left antrum when penicillin was injected intramuscularly in a dosage of 100,000 to 200,000 units per day for three weeks.

2. Treatment for an infection of a flat bone, including the frontal bone, is adequate when 200,000 units of penicillin is injected daily for three weeks,[1] so far as intramuscular injection of penicillin may aid in lessening the need for surgical intervention in this condition. In other words, an adequate trial of penicillin for the prevention or the treatment of osteomyelitis of a flat bone has been made when 200,000 units has been injected intramuscularly daily for three weeks. Of course, such a trial may be unsuccessful, with surgical intervention thereby becoming of utmost importance; in the present case osteoporosis of the walls of the left frontal sinus was controlled, and infection in that sinus cleared up.

3. Although it is desirable, it is not practicable to test the infective organism for penicillin sensitivity in every case. Such a test should, however, always be done if penicillin is employed in the presence of serious illness.

4. In this case, in previously normal sinuses of the right side acute maxillary sinusitis with ethmoiditis developed while 100,000 to 200,000 units of penicillin was being administered daily intramuscularly and after such treatment had been given for at least thirteen days.

REPORT OF A CASE

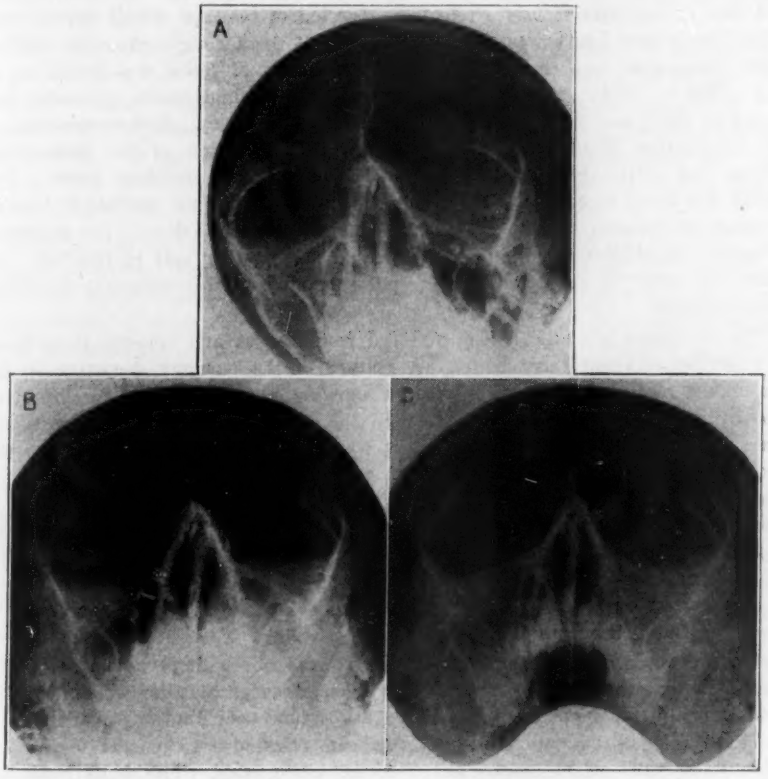
A 14 year old white school boy was first seen on Jan. 11, 1945, complaining of terrific pain in the left half of the forehead. He had had a cold in the head for sixteen days and a headache of varying severity over the left eye for ten days. The pain was at times so severe that it made him cry. He complained that the left side of his nose was blocked up and that a great deal of pus was discharging from his nose into his throat, although he blew his nose little.

Local examination of the nose showed very turgescent red nasal mucous membrane on the left with pinkish red thin nasal mucous membrane on the right. Thick gray creamy pus filled the upper half of the left nasal cavity as

From the Department of Otolaryngology, Massachusetts Eye and Ear Infirmary.

1. Keefer, C.: Personal communication to the author.

well as the left nasal meatuses. There was tenderness over the floor of the left frontal sinus with slight puffiness of the left upper eyelid. There was no tenderness over the left canine fossa. The left frontal sinus and the left antrum were dark on transillumination, while the right antrum and the right frontal sinus transilluminated clearly. The general physical and neurologic examinations showed nothing that was abnormal. A clinical diagnosis of acute pansinusitis of the left side, most marked in the left frontal sinus, was made.



A, first roentgenogram, made Jan. 11, 1945. It indicates pansinusitis of the left side, with pus in the frontal, ethmoid and maxillary sinuses. The front wall of the frontal sinus shows an area of marked porosity but no definite osteomyelitis. The outline of the frontal sinus is intact. There is definite thickening of the soft tissues overlying the frontal sinus.

B, roentgenogram taken February 2. Pus is present in the right antrum. The lining membrane of the right ethmoid cells is thickened. The left antrum shows a cyst. The frontal sinuses are clear.

C, roentgenogram taken February 12. The left frontal, ethmoid and maxillary sinuses have cleared except for the cyst on the floor of the left antrum. There is pus in the right antrum.

Roentgen examination of the sinuses was advised and was made immediately by Dr. A. S. Macmillan. His report follows: "There is definite pansinusitis of the left side with pus in the frontal sinus, the ethmoid cells and the antrum. There is an area of marked porosity of the front wall of the frontal sinus but

no definite osteomyelitis, and the outline of the frontal sinus is intact. There is definite thickening of the soft tissues overlying the frontal sinus." (Later roentgenograms showed porosity also of the posterior wall of the frontal sinus, detected by a special technic.)

The temperature was 100.2 F.; the pulse rate 90; the respiratory rate 20. The patient was admitted to the Massachusetts Eye and Ear Infirmary for treatment.

There he was given sulfadiazine with sodium bicarbonate. An initial dose of 4 Gm of sulfadiazine was given over a period of two and a half hours; then 1 Gm. every four hours was ordered. The usual therapeutic routine for sulfonamide compounds was employed; that is, the intake of fluids was forced to at least 3,500 cc. daily; the intake and the output of fluids were measured and recorded; the p_H of the urine was kept at 7.0 or higher. A determination of the sulfadiazine blood level, a blood count, an estimation of the hemoglobin content and a urinalysis all were ordered made every forty-eight hours. The patient was given a nasal spray, to shrink the tissues; compound tincture of benzoin by steam inhalations; codeine sulfate in ¼ grain (0.016 Gm.) doses when necessary for pain; pentobarbital sodium for sleeplessness, and other aids to comfort. An effort was made by nasal tamponage, spot suction and other measures to induce nasal drainage.

Twelve hours later the patient suffered from nausea and vomited three times in a period of about three hours. A culture of the purulent discharge in the left middle meatus had been reported as showing a coagulase-positive hemolytic type of Staphylococcus aureus. Although the sulfadiazine blood level was only 5 mg. per hundred cubic centimeters, it was decided to discontinue administration of this drug on the assumption that the vomiting was due to a sulfadiazine reaction. It was recognized, however, that incipient osteomyelitis might be producing extradural irritation either over the posterior wall of the frontal sinus or higher up on the squama of the frontal bone. There was no palpable edema of the forehead. The slight edema of the upper eyelid remained unchanged. It was recognized further that the toxicity of the infection itself might be a simple explanation for the vomiting.

On the other hand, nausea and vomiting were not without advantage to the patient in that this reaction provided an adequate argument for obtaining sufficient penicillin for further treatment. (At this time penicillin was available only for extremely ill patients and had not been released for general use.) The urine at the time of the nausea and vomiting was normal. The white blood cell count was 6,600 and the red blood cell count 3,400,000; the hemoglobin content was 86 per cent (Sahli). The administration of penicillin was started with a dose of 12,500 units, repeated every three hours. The nausea and vomiting rapidly subsided, and the patient improved clinically. He continued to have a profuse yellow mucopurulent discharge from the left middle and superior meatuses. His postnasal drip seemed to produce an irritating cough. The general physical examination and the roentgenogram of the chest showed normal conditions. After a week of penicillin therapy, roentgenograms of the sinuses were taken on January 18 and were interpreted by Dr. A. S. Macmillan as follows: "The left frontal, ethmoid and maxillary sinuses still are completely filled with pus. The bone is clean." Penicillin therapy was continued.

On January 25, further roentgenograms of the sinuses were interpreted by Dr. A. S. Macmillan as follows: "The lining membrane of the left frontal sinus is now only slightly thickened. The bone is still intact. The ethmoid cells are clear. The antrum contains thickened cystic membrane."

After a conference with Dr. Chester Keefer, it was decided to increase the penicillin dosage to 200,000 units daily for a third and final week of such therapy. Dr. Keefer stated that the reports to him from the country at large showed that with infection of flat bones the ideal dosage of penicillin had been found to be 200,000 units of penicillin daily for three weeks.

A day or two after the higher dosage of penicillin had been started, the patient began to complain of pain in the right half of the forehead and the right cheek and to have mucopurulent discharge in the right middle meatus, although the left side of the nose continued to improve.

With the administration of penicillin all signs and symptoms in the left side of the nose and face had cleared; that is, the mucous membrane, which had been red and swollen, shrinking poorly with adrenergic drugs (such as 0.25 per cent aqueous neosynephrine hydrochloride with a similar quantity of cocaine muriate), had lost its swelling entirely. The left middle meatus was staying open constantly with spraying of the nasal cavity with 0.1 per cent aqueous naphthyl methyl imidazoline (privine).

The clinical examination suggested the presence of right maxillary and ethmoid sinusitis. Roentgenograms of the sinuses were taken on February 2 and were reported on by Dr. Macmillan as follows: "Pus is present in the right antrum. There is a cyst in the left antrum. The lining membrane of the right ethmoid cells is thickened. The frontal sinuses are clear. The bone is clean." (See *B* in figure.)

A culture of pus taken from each middle meatus at this time was reported as follows: "Left nostril: coagulase-positive hemolytic Staphylococcus aureus. Right nostril: (pus from middle meatus), no growth." It was thought that the large dose of penicillin might be combating the growth of the organisms; so the bacteriologist by special methods attempted to rule out this possibility in further studies. Nevertheless, the culture remained sterile.

Two days later, after penicillin therapy was stopped, further material taken from each middle meatus was cultured and coagulase-positive hemolytic Staph. aureus was shown to be present on each side. Tests of this organism for penicillin sensitivity were then found to be as follows: With 1 unit of penicillin per cubic centimeter a 4 plus growth was obtained; with 5 units a 3 plus growth, with 10 units a 2 plus growth, with 25 units and 50 units no growth. The conclusion from these tests was that the staphylococcus strain being dealt with had the second highest penicillin resistance found in ordinary tests, which ordinarily go as high as 50 units of penicillin per cubic centimeter of culture medium.

The administration of penicillin was stopped because it seemed useless to continue it. There was a good clinical result in the left side of the nose and the left sinuses, but it had had no effect in large doses in preventing the development of right maxillary and ethmoid sinusitis.

Roentgenograms of the sinuses taken on February 12 were interpreted as follows: "The left frontal, ethmoid and maxillary sinuses have cleared except for the cyst on the floor of the left antrum. There is pus in the right antrum." (See *C* in figure.)

With the usual methods of simple conservative treatment, such as nasal sprayings, and local treatment designed to induce sinus drainage, the patient's infection on the right side improved satisfactorily. The further convalescence was uneventful.

COMMENT

The porosity of the anterior wall of the frontal sinus reported shown in the first set of roentgenograms was interpreted pathologically by me as vascular absorption of bone caused by dilated and engorged vessels in this location, probably with granulation tissue around the dilated vessels. Since the veins in the diploe of the anterior wall of the frontal sinus are in direct continuity with those of the squama of the frontal bone, it seemed entirely possible that spreading osteomyelitis might develop. In my experience penicillin therapy had been successful in controlling a large osteomyelitic area in the lower jaw of another patient, and it was considered desirable to utilize penicillin therapy for preventive purposes. Such therapy was successful.

The extraordinary fact, however, that a relatively sterile right maxillary and ethmoid sinusitis (*B* in figure) developed in spite of the penicillin that was being injected intramuscularly in a dosage of 100,000 to 200,000 units daily seemed to be of sufficient importance to be reported.

When this complication arose, it was assumed that an organism not affected by penicillin must be the etiologic agent and that some gram-negative organism would be found. The first surprise was that pus from the right middle meatus yielded a sterile culture while the penicillin therapy continued but that when this therapy was discontinued, a day later, it was possible to recover the same organism from the right middle meatus as had been found in the left middle meatus. The explanation appeared to be that this particular strain of coagulase-positive hemolytic Staph. aureus was itself resistant to penicillin.

Penicillin sensitivity is measured at the Massachusetts Eye and Ear Infirmary as follows: A high degree of sensitivity is evidenced by absence of growth when 1 unit of penicillin per cubic centimeter of culture is employed. The next step is the use of 5 units of penicillin per cubic centimeter; the next, 10, the next, 25, and finally, 50 units per cubic centimeter. The organism being dealt with in this case occupied the fourth place in this classification, in that 25 units per cubic centimeter was required to prevent its growth.

247 Commonwealth Avenue.

Clinical Notes; New Instruments and Technics

PLASTIC REPAIR OF A LARGE ALVEOLAR-ANTRAL FISTULA

MAJOR JOSEPH H. HERSH
Medical Corps, Army of the United States

e problem of repairing a large alveolar-antral of long standing is always difficult, and the on is pressed to select a procedure which will likely be successful in a given case. The many ions in operative procedure which have been pro- were reviewed by Ashley,[1] who in addition de- d a method which he favored. Recently Hill[2] ssed the matter again and suggested still another of operation. Of all the methods that have been bed, the latter is the only one that attempts to y the principles set down in plastic surgery for ring a defect in the wall of a body cavity—i. e., onstruction of a lining and a covering. This has a neglected aspect of the work probably because only of importance when the surgeon is dealing large defects.

the case reported here, the fistulous opening was rge and so long-standing that it was thought that ssful closure could be accomplished only if a pro- e was "plastically" correct. Hill's procedure was known at the time that the patient was under treat- In effect the method devised represents merely iation or extension of the technic which he out- Hill formed his lining by turning back a flap the gingival mucosa adjoining the fistula; then overed the raw surface with a sliding flap from buccal mucosa. This method, however, could not plied when the opening was large and teeth were nt near either limit of the opening, as in the pres- ase.

REPORT OF A CASE

31 year old Negro soldier presented himself with omplaint of recurrent left-sided headaches. These tarted eight years before and had become severe g the past year. He also complained of a bad in his mouth, which he ascribed to pus that leaked an opening in the left side of his upper jaw, over he wore a removable prosthesis which bore an ator that plugged into this opening. The opening been present for eleven years and followed the ction of three upper teeth. Unsuccessful attempts been made to close the fistula at the time that it oped, and then the obturator was devised.

amination of the nose revealed a high left septal tion and polyps in the left middle meatus. Trans- ination revealed darkening of the left frontal and ft maxillary sinus. In the left half of the upper teeth 5, 6 and 7 were missing, and there was a ous opening in the alveolar-gingival ridge which ured 13 by 5 mm. and was oval. The obturator into it permitted both air and pus to escape from the antrum when the patient forced air from the nose into the antrum.

Laboratory Reports.—The blood counts and the urine were normal. The Kahn test was positive. The Wassermann reaction of the spinal fluid was negative and the colloidal gold curve normal. A culture of the pus from the antrum revealed Streptococcus viridans and Haemophilus influenzae.

Roentgenograms. — The frontal sinus, the ethmoid labyrinth and the axillary sinus of the left side showed diffuse clouding. The sphenoid cells and all the structures of the right side were clear. Roentgen examination after iodized poppyseed oil 40 per cent had been instilled into the left maxillary sinus revealed only a small central pool of iodized oil.

Diagnosis.—The clinical diagnosis was left pansinusitis, left alveolar-antral fistula and latent syphilis.

Course. — Antisyphilitic treatment was started, and after six weeks of treatment with oxophenarsine hydrochloride, 0.06 Gm. biweekly, and bismuth subsalicylate, 0.26 Gm. weekly, it was felt that the patient was ready for a surgical procedure. The Caldwell-Luc operation and transantral ethmoidectomy were done on the left side. The antrum and the ethmoid cells were found to be filled with pale polypoid tissue and isolated pockets of pus. This tissue was all removed, with the pus, and a nasoantral window was made. The postoperative course was uneventful, and the healing was rapid.

The pathologist's report on the removed tissue was as follows: "The specimen consists of numerous strips of polypoid masses of pink edematous tissue.

"Microscopically, some of the pieces of tissue are partially covered by ciliated columnar epithelium. All of the connective tissue stroma is loose and edematous and shows marked infiltration with lymphocytes and plasma cells and moderate infiltration with polymorphonuclear neutrophilic and eosinophilic leukocytes."

The pathologic diagnosis was: polypi showing subacute and chronic inflammation.

During the patient's convalescence the left frontal sinus was irrigated and filled with a solution of a penicillin salt (20,000 units of penicillin) after each treatment. The sinus was free of pus after five such treatments.

Repair of Fistula.—On July 31, 1944 the alveolar-antral fistula was repaired in the following manner:

Step 1. A mucoperiosteal flap was raised (indicated as *A* in figure 1) from the buccal edge of the alveolar ridge toward the palate and from front to back so that the fistulous opening was incorporated in the flap. As the tissue was raised, as much of the lining of the fistula as possible was separated from the bony edges to remain attached to the flap. As usual, the bony defect was found to be much larger than the opening in the soft tissue indicated and was found to measure 20 by 8 mm.

1. Ashley, R. E.: A Method of Closing Antro- olar Fistula, Ann. Otol., Rhin. & Laryng. **48**:632 .) 1939.

2. Hill, F. T.: Management of Alveolar Fistula, Otolaryng. **40**:167 (Sept.) 1944.

Step 2. A buccal flap was elevated (indicated as *B* in figure 1). It was formed as a diverging flap to give it a broad base which would assure adequate nutrition.

Step 3. The buccal flap was then rotated in its longitudinal axis 180 degrees so that its epithelial surface was presented to the interior of the antrum and the raw surface was toward the oral cavity. The flap had to be formed large enough to permit this rotation without constriction of its blood supply and large enough to cover the defect. It was then sewed into place with the fine silk as indicated in figure 2.

Step 4. The edges of the fistulous opening in flap *A* were then freshened and trimmed straight to permit their approximation with fine silk sutures. Flap *A* was then sutured into place with interrupted silk sutures so that its raw surface was against the raw surface of flap *B* as shown in figure 3. A piece of stent was molded to cover all of flap *A* and then wired with moderate pressure as a cover by figure-of-eight l to the adjacent teeth.

Healing was uneventful. The sutures were re on the fifth day and the pressure dressing (stent was used until the tenth day.

Step 5. At the end of the third week the brid tissue created by the buccal flap *B* was cut thr near the outer edge of the alveolar ridge. The pe was then rotated back to its original position, granulating surfaces were freshened and the flap sutured into position to recreate a somewhat shall but definite gingivobuccal sulcus.

Fig. 1.—The lines of incision and the formation of the flaps are shown.

Fig. 2.—Flap *B* has been rotated 180 degrees and then pulled over to cover the bony defect. It has moved into place and sutured to the edges of the defect made by the elevation of flap *A* and to the perios of the attached edge of flap *A*.

Fig. 3.—The edges of the fistulous opening in flap *A* have been trimmed straight and approximated fine silk sutures. Flap *A* has then been fixed in place overlying flap *B*.

SUMMARY

The method of repair of a large alveolar-a fistula described provides both a covering and a li It is believed that such a procedure, when one is ing with large defects, assures a successful re reduces to a minimum the raw surfaces exposed i mouth and does not result in complete obliterati the gingivobuccal sulcus.

OTOSCLEROSIS

An Index of the Literature with Abstracts and an Index of Authors According to Subjects for 1943

THIS is the first yearly installment of "Otosclerosis," Volume IV, to be issued by the tral Bureau of Research of the American logical Society, Inc.[1]

With a view to a wider distribution, the CHIVES OF OTOLARYNGOLOGY is publishing text in the February issue, and it is hoped we may obtain this cooperation for many rs to come, to the best interests of all con- ned.

During the war there has been difficulty in ining the usual number of publications, and e is a lag in receiving many foreign articles, this will be made up from time to time, so eventually all may be printed in subsequent rs for placement in the volume IV binder.

The factors underlying heredity, causation, y diagnosis, pathologic aspects, arrest, reces- or cure of otospongiosis are still largely nown, but many pertinent facts are accumu- g. The outlook for prevention or arrest of disorder is not as hopeless as it has formerly med.

t is our intention in the future to include e extensive abstracts of selected articles than he past. No endorsement or condemnation implied by these choices. The Editor has ven to include in the index all literature con- ning facts or hypotheses pertaining to oto- rosis and to set forth the opinions of the hors quoted without comment.

As in prior installments, all titles are given in English and the names of all media of publication in the language of the original. Dr. Franz Altmann has prepared the translations and outlined the abstracts for 1943. The next installment (1944 and 1945) will be published in the fall of 1946, and subsequent installments at intervals of one year.

A few copies of volumes I and II, 1928, volume III, 1936, and volume IV, 1942, with and without binder, are still available.

This installment, complete with Title page and with Index of Authors according to subjects and properly perforated for the loose leaf binder (volume IV of "Otosclerosis"), may be obtained at cost plus shipping charges ($1.00) from

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INDEX OF LITERATURE WITH ABSTRACTS

1943

Anson, B. J., and Cauldwell, E. W. Stapes, Fissula Ante Fenestram and Associated Structures in Man: IV. From Fetuses 75 to 150 mm. in Length, *Arch. Otolaryng.* **37:** 650 (May) 1943.

The conclusions offered by Anson and Cauldwell are as follows:

In the 75 and 100 mm. stages, the stapes attains a form which, in general, is retained throughout the remaining chondral phase of its development; a cylindric capital portion and a platelike basal part are recognizable. The head becomes elongate and foveate and the base circumferentially lipped, but the crura remain columnar, round or oval in cross section. In the succeeding series, and up to the time ossification begins, the chief change in the stapes is in the direction of growth; the cellular structure remains fundamentally the same; the dominant theme is the amplification of an established pattern. In the 150 mm. fetus, the primary center of ossification has appeared on the obturator surface of the base. It is a remarkable fact that at the time osseous change is first observed the stapes is actually more massive than it is in any subsequent stage, either fetal or postnatal, and its dimensions are about as great as they will ever be. Later, in the 183 mm. fetus, when ossification has progressed to the point at which the crura and base are enveloped by periosteal bone, the form is still substantially that of the stapes of the 100, 111 and 150 mm. fetuses. Only after bone,

[1] The Central Bureau of Research of the American logical Society, Inc., is constituted as follows: rd of Trustees of the Research Fund: D. Harold lker, M.D., chairman, Boston; Wesley C. Bowers, D., New York; Edmund Prince Fowler, M.D., New rk; Marvin F. Jones, M.D., New York; John R. e, M.D., New York; J. Gordon Wilson, M.D., cago; D. E. Staunton Wishart, M.D., Toronto, Can- Lay Advisers of the Board: George E. Coleman, ta Barbara, Calif.; E. Ericson, New York; Samuel d, New York. Consultants to the Board: Madi- Bentley, Ph.D., Sage professor of psychology, Cor- University; J. B. Collip, M.D., D.Sc., F.R.C.P., .S., professor of biochemistry, McGill University; y R. Guild, Baltimore; W. A. Neilson, Ph.D., D., L.H.D., Litt.D., Falls Village, Conn.; Oscar dle, Ph.D., Department of Genetics, Carnegie Insti- on of Washington. Secretary: Edmund P. Fowler, D. Treasurer: Walter C. Baker. Depositary: ranty Trust Company of New York.

once laid down, has been partially removed does the stapes assume the channeled form which characterizes the "adult" ossicle.

The crura are first set off from the base and head in the 34 mm. fetus. Crural growth is appreciable in succeeding series; the columns not only lengthen but become separately distinguishable parts through their relation to the broadening base: They seem to be implanted in a plate the margins of which extend beyond the areas where the crura and base become continuous. With onset of ossification in the stapedial base, the posterior crus is invariably the first to be involved.

In the 100 and 111 mm. stages the capital extremity is cylindric, deeply foveate on its lateral (incudal) surface and widely mergent with medial crural extensions; an ill defined neck now makes its appearance, both the head and the neck approaching their definitive external shape, although they are continuous parts of a solid, cartilaginous cylinder.

As soon as the process of ossification spreads to the crura and neck, growth of the stapes ceases; thereby, in the 183 mm. fetus, is produced a continuous tube of bone of modified annular form, cartilage remaining as a basal plate and a capital cylinder. Once formed in bone, subsequent changes are of a unique order: All periosteal bone facing the obturator foramen is destroyed, and together with it the transitory endochondral bone and associated marrow which originally were contained within the neck, crura and head. Through the gradual spread of tympanic mucous membrane, the submucosal tissue comes to occupy these excavated portions of the ossicle.

The tissue of the annular ligament does not differentiate with equal rapidity over its several portions; its development seems to be consistently retarded on the inferior stapedial margin; on other margins it consists of dense, radially directed fibroblastic tissue, in all stages beyond that of 126 mm.

The vestibular (oval) window is a well defined hiatus conforming closely to the outline of the stapedial base (foot plate) in the 75 and 100 mm. fetuses. The shape of the window is apparently altered by the upward growth of the promontory (111 mm.), by stapedial overlapping of the fenestral margin (112 mm.) or by marginal overlapping of the stapedial base (anterior extremity of base, 150 mm.). The window approaches its adult dimensions in the 150 mm. fetus; changes toward the adult form are practically complete as early as the 183 mm. stage. The fenestral margin is never completely ossified; the articular edge remains a rim of cartilage throughout life.

The fissula ante fenestrum makes its appearance in the 34 mm. fetus; in later stages, exclusive of the 150 mm. stage, a series of regressive and progressive changes in its constituent tissues mark this area as one of histologic instability. A tympanic orifice is vaguely suggested in the 50 mm. fetus, but is well established in the 75 mm. fetus and in succeeding stages. Variations in fissular shape and extent are multifold: An intracartilaginous seam may extend from the tympanic to the vestibular surface; an auxiliary or fenestral orifice may appear; a deep wide cleft may pass from the vestibule, through the anterior fenestral margin and onto the tympanic surface of the labyrinth. In every specimen studied the tympanic orifice of the fissula is located more superiorly than the vestibular; the latter is regularly the larger of the two and the closer to the fenestral margin.

The fossula post fenestram is an inconstant channel in the capsular wall situated posterior to the vestibular window, as its name implies; it is present in the and 147 mm. fetuses and in both communicates with the vestibular cavity.

Benesi, O. Some Factors Involved in the velopment of Otosclerosis, *Laryngoscope* 343 (May) 1943.

Otosclerosis is an abnormality in the deve ment of the otic capsule. Normally the capsule does not change after birth and tains in its enchondral layer immature throughout life. At an early stage in otos rotic foci one finds many newly formed mar spaces and engorged blood vessels, while immature tissue has disappeared to be repla later by newly formed, irregular bone. S the transformation of cartilage into bone depe on the functioning of blood vessels, one assume that in the zones where cartilage isla are normally present at birth blood vessels h been obliterated and therefore these cartil rests have lost their capacity to be transfor into bone. It seems reasonable that the s forces which normally are concerned with b formation may act on this immature tis under certain conditions in later life. It is p sible that in some persons certain of the num ous blood vessels which normally are obliter fail to undergo obliteration and continue transform the cartilaginous elements into bo The ultimate result of this process would be complete ossification of the otic capsule, a co dition which actually is observed in some ca of otosclerosis.

On this basis the author believes that otosc rosis may be regarded as a continuation growth beyond normal limits. Slow at the ons the otosclerotic process is accelerated by introduction of secondary factors, such as p berty, pregnancy, puerperium, violent exerci conditions which are accompanied by high te peratures and disturbed general metabolism.

Benesi, O. A Case of Unilateral Otoscleros with a Microscopic Description of the Foc *Laryngoscope* **53**: 17 (Jan.) 1943.

The author reports the histologic examinati of a large otosclerotic focus situated in front the oval window in a man 52 years of ag There was no osseous fixation of the footpl of the stapes. The other temporal bone w free from otosclerotic changes. The aut assumes that the cause of the development an otosclerotic focus is related to the abnorm presence of blood vessels at the site of orig The focus appears to pass through a phase relative inactivity and then the changes act

gradually in response to a physiologic or logic stimulus involving the general circu- system.

el, W. H.; Ghormley, R. K., and **Camp, D.** Osteogenesis Imperfecta, *Radiology* 0:145 (Feb.) 1943.

teogenesis imperfecta is an anomaly in h the etiolgic factor is probably some defect ent in the germ plasm. This defect may ereditary or sporadic. The result is a mal- lopment of mesenchymal tissues of the , which is manifest also in brittle bones, scleras and otosclerosis. Otosclerosis oc- d five times in 11 cases of the hereditary and five times in 29 cases of the non- ditary type. The higher incidence in the er group can be explained no doubt by the that those patients lived to a greater age the otosclerosis had more time to develop. scleras were present in 10 of the 11 heredi- cases and in 21 of the 29 nonhereditary .

nner, H. Otosclerosis, *Laryngoscope* 53: 736 (Nov.) 1943.

runner reports his experiences based on the cal and pathologic examination of 17 cases the clinical examination of 1 case.

linically women may be more frequently ted than men, but histologic examination s that foci are found with approximately same frequency in both sexes.

mong the 17 patients there was diminished ing in 12 (8 women and 4 men). Eight ed chronic diseases, such as syphilis of the d vessels, chronic suppuration or catarrh of middle ear or tumor of the brain, any of h might impair the hearing without otoscle- s. In 2 patients ankylosis of the stapes was nd. In 1 patient there was severe congenital formation of the cochlea; in another, "simple phy of the cochlea." The author observed ylosis of the stapes in only 17.8 per cent of patients but surmises that it is actually more uent. If the ankylosis is of small extent, it be overlooked unless every serial section is ned and studied.

n 4 of the 13 cases in which both temporal es were examined the pathologic process was lateral. In none did the otosclerosis produce ical symptoms. In 12 cases with complete , 11 were without a history of tinnitus. The hor feels that neither ankylosis of the stapes atrophy of the cochlea necessarily produces itus, at least not permanent tinnitus.

The question of the effect of pregnancy on otosclerosis is a complex one, and it is better to avoid general statements concerning the relation of the two conditions.

Chiarino, A. New Trend Toward Etiology and Pathogenesis of Otosclerosis: Poradenolymphitis and Otosclerosis, *Rev. argent. dermatosif.* **26:** 537, 1942.

Ten of 15 patients suffering from otosclerosis showed diminution of corneal sensibility. In 10 of the patients there was abnormal visibility of nerve fibers in the central part of the cornea; in 2, hypotension of the bulbus, and in 3, congestion of the veins in the fundus. In all 15 patients the intracutaneous Frei test was positive. In all the 6 patients in whom cisternal puncture was done, an increase in the albumin content of the cerebrospinal fluid was noted. The author believes that an etiologic connection exists between otosclerosis and poradenolymphitis (granuloma inguinale) of extragenital origin.

Fabricant, N. D. Surgical Treatment of Otosclerosis, *Am. J. M. Sc.* **205:** 604 (April) 1943.

This is a review of the problem.

Farber, J. E., and **Margulis, A. E.** Blue Scleras, Brittle Bones and Deafness: Report of an Afflicted Family, *Arch. Int. Med.* **71:** 658 (May) 1943.

The authors report the results of their examination of a family of 52 members and give a genealogic tree.

Twelve members had the blue sclera syndrome. Seven of the 12 members had both blue scleras and brittle bones. Four suffered not only from these defects but from deafness. One so far shows only blue scleras. Eight of the affected members are males; 4 are females. The condition is transmitted from generation to generation. Transmission was by a male parent in 2 instances and by a female parent in 2 instances. The inherited factor appears to be a mendelian dominant, not sex linked. The exact nature of the anomaly is unknown. The established dictum that unless blue scleras are present the other features of the syndrome do not appear is illustrated in this series. In 1 patient otosclerosis with ankylosis of the stapes was probably present. The tendency of the bones to fracture is a recurring or cyclic affair; it diminishes with age in spite of the generalized osteoporosis. The parathyroid glands are apparently not implicated pathogenetically.

Fleischmann, L. Contributions to the Surgical Treatment of Otosclerosis, *Acta oto-laryng.* **30:** 139, 1942.

A modification of Holmgren's method is described. The first step is an incomplete mastoidectomy with wide opening of the antrum and exposure of the lateral bony semicircular canal. A retroauricular incision and local anesthesia are employed. The posterior wall of the osseous part of the external auditory meatus is removed with the exception of the medial end, where a 5 mm. wide bridge is preserved. A flap is then made from the skin of the posterior wall of the canal with its base at the bony bridge just mentioned. This flap can be turned around the bridge so that it covers the prominence of the lateral semicircular canal. The last step is the fenestration of the lateral semicircular canal. This is done with curets, gouges and chisels. The window is made away from the ampullar end and on the peripheral surface of the lateral canal, where the membranous canal is farther away from the osseous wall than on the anterior surface. The author follows the advice of Engström, who claimed that the fistula should not be made near the ampullar end because of the danger that newly formed bone could grow into the ampulla and damage the sensory epithelium. The endosteum of the canal is left intact. The fenestra is covered with a thin leaf of gold or silver and then covered with the flap. The cavity is filled with vioform gauze through the external meatus, and the retroauricular wound is closed. The epidermization of the operative cavity takes four to five weeks.

The operation was performed on 14 patients; in some of them there was advanced degeneration of the nerve. After the operation the hearing was considerably improved, particularly in those for whom the operation was indicated, but later the improvement was partially lost.

Fowler, E. P. The Control of Head Noises: Their Illusions of Loudness and Timbre, *Arch. Otolaryng.* **37:** 391 (March) 1943.

Two types of tinnitus are distinguished — vibratory and nonvibratory — the first being mechanical in origin, the second biochemical in origin. If caused by irritation of the auditory neural elements, "it is an illusion of sound." The author shows how the loudness of tinnitus may be measured and its wave frequency approximated. It was found that although a patient may complain of intensive tinnitus, it is often only 5 or 10 decibels above threshold in loudness. The author uses this objective method in aiding the patient to understand better the severity of his annoyance and so not to overestimate it. stances are given of the different intensities nec sary to mask tinnitus, and these differences the author's opinion, are due not only to the l ness of the tinnitus but to its point of origi i. e., in the peripheral apparatus in the cochle in the central tracts or nuclei. Different type tinnitus are discussed but "the presence of nitus is always associated with more or deafness." Suggestions as to management treatment of tinnitus both in young and in o people are given, and the conditions (includ otosclerosis) commonly associated with the l sion of sound called "nonvibratory tinnitus" discussed.

Guggenheim, L. Therapy of Deafness: Rep of Cases, *Laryngoscope* **53:** 441 (July); (Aug.); 571 (Sept.); 653 (Oct.) 194

The prevention of deafness from otosclerosis stressed. The methods in order of importa are: 1. Discourage propagation by persons w have otosclerosis. 2. Discourage marriage b tween two otosclerotic persons. 3. Diagnose t condition early in order to forestall as far possible enlargement of the focus and neuropat In treating otosclerosis there are three disti aims: 1. Prevention of spread of the proc from the focus of dystrophic bone. In t author's opinion this can be achieved by a pro diet with added calcium, and phosphorus (calcium phosphate) and vitamins. 2. Reversal the neural injury resulting from pressure the ramus cochlearis at the internal meatus a from the ingrowth of dystrophic bone in t region of the round window, and other as y unknown factors. According to the author, th is achieved with injections of vitamin B compl and insulin, combined with oral intake of vitam B complex. 3. Alteration of cerebral meta olism, with improvement in the perception a the interpretation of sounds.

Guggenheim, P., and **Guggenheim, L.** Bo Fistula: Further Studies, *Arch. Otolary* **38:** 139 (Aug.) 1943.

Despite the fact that the fistula operation today being performed with enthusiasm by few otologists, it is pointed out that no one h yet succeeded in keeping patent an artifici fistula in an experimental animal with any meth which could be employed safely in the wall the human labyrinth.

An attack on this problem was developed fro biochemical considerations. An attempt w made to inhibit chemically the regeneration the bone within experimental fistulas made

kulls of rabbits. The copper ion was believed ising by virtue of its catalytic oxidizing on vitamin C, which is essential to the for- on of the matrix of bone. Trichloroacetic and silver nitrate solution were used as rols.

pper sulfate produces much fibrosis and s bone regeneration. Silver nitrate pro- much fibrosis and successfully inhibits bone eration. Trichloroacetic acid, the most ic, produces no fibrosis and completely in- s bone regeneration. Prevention of osteo- sis can be achieved by the use of a suf- tly powerful caustic, but specific interfer- with the biochemical mechanism of bone ation by copper is not demonstrated. The ic agents which successfully prevent osteo- sis in experimental fistulas do not appear to uitable for application to the labyrinth.

ler, R. P. Audiometric and Word Test Findings: Preliminary Report, *Ann. Otol., Rhin. & Laryng.* **52:** 25 (March) 1943.

here is a high correlation between audio- ic and word test scores. A lowering of the shold of hearing at any point results in a ring of the word test score. The word test e of a person with high tone losses is often ed, while the same person may give 100 ent on a number test. This observation is y explained by the fact that the understanding mbers depends largely on the differentiation wel sounds involving the middle frequencies, e the understanding of words other than bers calls for differentiation of consonant ds by accurate hearing in the upper tonal e of speech.

otosclerosis the audiometric findings are al of conduction deafness in general, and diagnosis must be based on the combined vledge obtained from the history, the physi- examination and the study of the hearing.

here are three principal types of curves: (1) rizontal lowering of the audiometric curve ghout the tone range, (2) a greater loss in ow tone range, (3) a greater loss in the high range (most frequent).

the air conduction curve drops, the bone uction curve may drop for the higher fre- cies; or it may remain nearly normal in the nce of marked lowering of the air conduc- curve.

afness of otosclerotic origin often progresses ly in children 11 to 13 years old and occa- lly in those 9 years old. In children with lerotic backgrounds, all types of slight devia- from normal hearing have been found.

Kelemen, G. Otosclerotic Focus Outside the Inner Ear Capsule, *Laryngoscope* **53:** 528 (Aug.) 1943.

In the osseous septum between two pneumatic cells in the immediate vicinity of the cortex of the tegmen antri, changes were found in a circumscribed area which histologically were similar to those encountered in otosclerosis.

Kelemen, G. Malformation Involving External, Middle and Internal Ear, with Otosclerotic Focus, *Arch. Otolaryng.* **37:** 183 (Feb.) 1943.

Kelemen gives the results of the histologic examination of serial sections of a temporal bone from a 10 year old girl. There was a malformation of the external, the middle and the internal ear. Near the lateral border of the footplate of the stapes there was "a small focus of reticulation representing the earliest stage of otosclerotic change."

Lewy, A., and **Leshin, N.** Functional Examination of Hearing, *Arch. Otolaryng.* **37:** 82 (Jan.); 242 (Feb.) 1943.

This is a critical review of the literature of 1941 and 1942.

de Lima, E. Surgical Treatment of Otosclerosis, *Rev. brasil. de cir.* **12:** 247 (March) 1943; *Bol. Col. brasil. de cirurgiões* **18:** 151 (July) 1943.

De Lima discusses the disease from the point of view of histopathology and considers the various methods of surgical treatment, particularly Lempert's modification of 1938. He reports the results observed in 2 patients who were operated on; in one the hearing was unchanged, and in the other it was slightly improved five months after the operation.

de Lima, E. Anatomicosurgical Study of a New Route of Access in Surgical Therapy of Otosclerosis, *Rev. med.-cir. do Brasil* **15:** 101 (Jan.-March) 1943.

This is a description of Lempert's nov-ovalis technic.

Mueller, W. Otoscopic Picture in Deafness, *Ann. Otol., Rhin. & Laryng.* **52:** 20 (March) 1943.

Mueller's article is based on examination of 689 persons. The otoscopic picture in health and in deafness is given. The data obtained aid greatly in the classification of patients with idiopathic deafness. The appearance of the ear

drum may at times tell nothing as to the type or the severity of the hearing loss. This is often so in uncomplicated otosclerosis.

Nilsson, G. Some Aspects of the Differential Diagnosis of Obstructive and Neural Deafness, *Acta oto-laryng.* **30:** 125, 1942.

The author's summary is substantially as follows:

From the point of view of the differential diagnosis of neural deafness the tuning fork tests are not always adequate and are, in fact, often misleading. Furthermore, audiometric investigations disclose certain sources of error, the most important of which is cross-conduction to the other ear in tests employing bone conduction. Through a careful investigation, with and without application of noise to the ear not subjected to examination, a reliable diagnosis will, however, be arrived at in most cases.

The only safe method is to examine the aural perception of sounds whose intensity is above threshold value. In the case of neural deafness Fowler's "recruitment phenomenon" will be produced. This does not occur with defects of conductivity. The "recruitment phenomenon" is demonstrated by means of "balance tests." Finally, an account is given of Lorente de No's explanations of the recruitment phenomenon.

Perlman, H. B. Lesions of the Conduction Apparatus, *Arch. Otolaryng.* **37:** 680 (May) 1943.

If the mass or the resistance or damping of the conducting mechanism is increased, its response to high frequencies is affected first. This is seen experimentally by loading the anteroinferior part of the drum with a few drops of water, glycerin or mercury and clinically when a hemorrhagic blister or a large granuloma is present on the outer surface of the drum or when fluid presses against the inner surface of the drum, as in exudative catarrhal or suppurative conditions in the middle ear.

If the tension or stiffness of the conducting mechanism is increased, the mass and the damping remaining unchanged, the mechanism's response to the low tones is interfered with first. This is seen when there is inequality between the air pressures on the two sides of the drum membrane (rapid descent in an airplane, driving through the mountains, change of "altitude" in the pressure chamber, tubal obstruction). A small focus of infection in the annular ligament of the stapes causes a slight increase in stiffness of the conducting system and thus a loss of low tones. Complete immobilization of the annular ligament increases the stiffness so markedly that the hearing for air-conducted sounds is uniformly reduced. If both windows are closed with bone or experimentally with plaster of paris, this completely abolishes hearing by air conduction as well as that by bone conduction, even though end organ is normal. Closure of both win with a soft substance leaves hearing acuity bone conduction intact but reduces that fo conduction.

Portmann, G. Considerations on the Pa genesis of Progressive Deafness, *Presse* **50:** 674 (Nov. 7) 1942.

What is progressive deafness to the Amer authors is identical with what is otoscleros the German and otospongiosis to the Fr authors. There is a close relationship bet the localization of the otosclerotic foci and arrangement of the vessels in the capsule. author thinks that local vascular disturba (congestion or venous stasis) could be res sible for the development of the foci. E lymphatic hypertension could bring about promote the establishment of the vascular nomena which eventually lead to the deve ment of the otosclerotic foci. It could also plain the slowly progressive character of deafness, which could not be explained by fixa of the stapes alone. The latter would resu a definite but not a progressive amount of ness. The author believes that decompressio the labyrinth by fenestration relieves the e lymphatic hypertension and arrests the prog of the disease as long as the labyrinthine fis remains patent.

Richardson, J. R. Anatomy and Physiol of the Ear, *Arch. Otolaryng.* **37:** 567 (A 1943.

This is a critical review of the available li ture from November 1941 to December 1

Rosenberger, H. C. Otosclerosis in Iden Twins, *Arch. Otolaryng.* **38:** 594 (D 1943.

Rosenberger records the history of otoscle occurring in identical twin girls and gives t audiograms taken at the ages of 13, 16 an years. So far as is known, the parents did show clinical otosclerosis. The audiograms very similar and showed a gradually increa loss of hearing, more marked with the high quencies. There were many infections o upper respiratory tract and a progressive of hearing with every infection. The low t (128 and 256) as well as the high (8192 9747) showed very slight if any increase of ness during the four years of observation. hearing for bone-conducted sound went more rapidly with high than with low ton

...e, R. Treatment of Progressive Deafness, *Rev. brasil. de oto-rino-laring.* **10:** 647 (Nov.-Dec.) 1942.

...he author reviews the treatment of chronic ...rrhal otitis media, otosclerosis, nerve deaf- ... and deafness from retrolabyrinthine lesions.

...mbaugh, G. E., Jr. Deafness or Impaired Hearing, *J. Michigan M. Soc.* **42:** 525 (July) 1943.

...asing his judgment on experience gained in ... hundred and twenty-six fenestrations, the ...or states that an improvement of hearing ...ntained for longer than six months may be ...rded as probably permanent. In from 1 to 2 ...cent of his cases the hearing was worse after ...peration, in 6 to 8 per cent the improvement ... only temporary, but in the remaining 90 to ...er cent there was a permanent improvement ...0 to 47 decibels above the preoperative level. ...re were no fatalities and no serious compli- ...ons. The ideal candidate for the operation ...a 50 to 60 decibel loss for the speech tones ... normal hearing for bone conduction, but ...th while improvements can sometimes be ...ined in those with 80 to 90 decibel losses.

...mbaugh, G. E., Jr., and Wojniak, F. Chronic Progressive Deafness, Including Otosclerosis and Diseases of the Inner Ear, *Arch. Otolaryng.* **37:** 856 (June) 1943.

...his is a critical review of the literature of ...l.

...th, J. M. The Fenestration Operation for Otosclerosis: Report of Thirty-Two Cases, *Arch. Otolaryng.* **37:** 174 (Feb.) 1943.

...ighteen patients were operated on with Lem- ...'s original method and 14 with Lempert's ... method, the "fenestra nov-ovalis" technic. ...ost of the operations the endaural approach ... used. A number of good results, with im- ...ement of the hearing to within satisfactory ...nsities for the conversational speech range, ...e obtained with the original method, and the ...portion of successful operations has been ma- ...lly increased by the new method. The longest ...od in which the improvement was maintained ...e time of publication was two years, but the ...or feels that if the improvement is maintained ...eight months, it will be lasting. In 3 of the ... 18 subjects a revision was carried out and ...istula found closed with newly formed bone. ... bone was removed, and in 1 instance the ...la remained open thereafter. The operation ...ld not be carried out unless the bone con- ...ion loss is less than 25 decibels; preferably this loss should be no more than 20 decibels for frequencies of 512, 1024 and 2048. The drum membrane should be intact and the eustachian tube patent. The oldest patient operated on was 49 and the youngest 14 years of age. The age of the patient does not influence the result, provided the bone conduction is satisfactory.

With proper asepsis and technic, the fenestration operation can be performed without undue risk. No facial paralysis, endocranial complication or diffuse labyrinthitis was noted. The hearing in the patients in whom the fistula closed was not made worse; it merely receded to its preoperative level.

Smith, K. R. Bone Conduction During Experimental Fixation of the Stapes, *J. Exper. Psychol.* **33:** 96 (Aug.) 1943.

Experiments were made on cats (stapedial motion relative to the skull was restricted in 15 series of tests in 8 ears) to determine whether or not stapedial motion is important in the response of the ear to bone-conducted sound. The positive results appear to support the inertia theory of bone conduction elicitation and suggest that modification of stapedial motion might be involved in the causation of some forms of bone conduction deafness, especially otosclerosis. It was found that fixation of the stapes relative to the skull, when sufficient to reduce profoundly the synchronous cochlear responses to air conduction, also substantially reduced responses to bone conduction in the middle frequencies. Stapedial motion relative to the skull is important in the response of the cat's inner ear to bone-conducted tones of middle frequency. The investigation indicates the clinical importance of lesions of the middle ear in the causation of bone conduction deafness. An explanation of the fact that the ossicular system is important for bone conduction only within the middle range of frequencies is suggested in terms of the presence and influence of the resonant frequency of the ear. In the cat the latter is probably within the middle range of frequencies, from 500 to 1500; in human beings it is approximately in the same range or slightly higher.

Spira, L. Etiology of Otosclerosis, *J. Laryng. & Otol.* **58:** 151 (April) 1943.

According to the author, otosclerotic foci are closely similar in appearance to marbled teeth and mottled nails, both of which are known to be produced by protracted action of fluorine. Moreover, several signs and symptoms accompanying otosclerosis are identical with those constituting the disease picture of chronic fluorine poisoning.

The author suggests that otosclerosis is a manifestation of fluorosis, and that it can therefore be prevented.

Sullivan, J. A. Surgery of the Mastoid, *Arch. Otolaryng.* **37**: 845 (June) 1943.

Encouraging results were observed in 75 patients operated on in accordance with Lempert's technics. The one stage fenestration offers, for the first time, a means of restoring hearing in patients with otosclerosis. It is indicated for those whose hearing loss is sufficient to constitute a handicap and who have an intact nerve of hearing for the conversational frequencies. It is technically intricate, delicate and time consuming, but if the procedure laid down is correctly followed at each successive step, a practical gain in hearing will be secured.

Tato, J. M. Surgical Therapy of Otosclerosis, *Rev. otorrinolaring.* **3**: 88 (Sept.) 1943.

The problem of otosclerosis is reviewed in great detail, and particular attention is paid to the question of operative treatment. The operations can be divided in two groups. Those of the first group attempt to eliminate a presumed causative factor: unilateral parathyroidectomy or ligation of the inferior thyroid arteries (Alonso and Chiarino) or section of the vein accompa ing the greater superficial petrosal nerve (W maack). Those of the second group try to prove the function without influencing the ca plugging of the round window niche (Hughs or by creation of a new window opening into labyrinth (Holmgren; Sourdille; Lempert).

The author operated on 2 patients after W maack's method without success and on 2 patie after the method of Alonso and Chiarino w moderate success in 1. In a patient operated after Hughson's method there was an impro ment of 10 decibels for the frequencies betw 256 and 2048 after one year. Thirteen patie were operated on with the original technic Sourdille, 20 with the Sourdille-Lempert stage technic, 2 with Holmgren's one stage te nic, and 6 with Lempert's fenestra nov-ov technic. In 16 per cent of the ears operated there was marked improvement, with restorat of "practical" hearing lasting more than months; in 26 per cent there was improvem but they had been recently operated on. In per cent the improvement was only slight, 23 per cent there was no change and in 12 cent the hearing deteriorated. The author f that the results will be better with further provement of the technic.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

ADVANCES IN THE UNDERSTANDING OF RHINOLOGIC AND OTOLOGIC CONDITIONS RELATED TO THE NERVOUS SYSTEM

A Critical Survey of Recent Literature

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NEWARK, N. J.

IT IS my hope that in the following selections from the recent literature my fellow otorhinologists will find some assistance in understanding general diseases of the nervous system as expressed by aural and nasal manifestations. For it is my contention that otorhinology has passed from being a specialty exclusively concerned with infections and diseases of the ear and nose to one which comprehends also an important domain of general medical diagnosis. It is from this point of view that the present survey has been made.

I have tried to put into my headings such anatomic, diagnostic and clinical leads as may be of service to the neuro-otologist. These are often apparently irrelevant to the subject discussed in the article reviewed.

An index of (1) the headings, (2) the subjects and (3) the authors is now in preparation and will be furnished on application, so that it can be added to "Eagleton's Index, 1922-1940."

BIOGRAPHIES OF WILDE AND MACEWEN

Two recent biographies—"Victorian Doctor: The Life of Sir William Wilde,"[1] and "The Life and Teaching of Sir William Macewen"—contribute to otologic history.

Wilde opened St. Mark's, in Dublin, Ireland, as a "Hospital and Dispensary for Diseases of the Eye and Ear" in February 1844. For a long time it was the only institution which provided systematic teaching on diseases of the ear and on aural surgery. Postgraduate students flocked to it from all over the world, "particularly from America." Wilde claimed, justifiably, that his statistics from 1844-1845 on were the earliest complete records of diseases of the ear published.

1. Wilson, T. G.: Victorian Doctor: The Life of Sir William Wilde, London, Methuen & Co., Ltd., 1942, p. 211.

Wilde's reputation as an otologic leader was assured by his book "Practical Observations on Aural Surgery and the Nature and Treatment of Diseases of the Ear," published in 1853. It was in reality the first modern textbook on aural surgery. An American edition was prepared by Dr. Addinell Hewson, who had been a pupil of Wilde's. "The clinical case reports . . . are models of clear reporting. In reading them one is almost transported to the bedside of the patient" (Dr. Douglas Guthrie [2]).

In "The Life and Teaching of Sir William Macewen," Bowman,[3] Macewen's biographer, comments on the stupendous events which occurred in Glasgow, Scotland, between the years 1860 and 1900, "for it was in this city that the most significant medical revolution since the time of William Harvey took place."

Ballance [4] remarked: "Sir William Macewen lived in a great age, when figures such as Virchow, Pasteur, Lister, Helmholtz, Kelvin, Michael Foster, Hughlings-Jackson and David Ferrier were actively at work."

To Macewen belongs the distinction of having been the chief pioneer in craniocerebral surgery. He has been called "the father of brain surgery," for his early work antedated by several years that of Victor Horsley (Percy Sargent). Bowman gives the following account of Macewen's surgical acumen:

> In the year 1876, a boy of 11 was admitted (Glasgow), . . . and Macewen made a diagnosis of a focal cerebral lesion, presumably a pyogenic abscess. He narrowed . . . its location to a point situated between the center for speech and the internal capsule in Broca's convolution. . . . He proposed to open the skull and to . . . evacuate the abscess; but he was unable to obtain consent. . . .
>
> After the child's death he performed the operation on the dead body . . . as he would have done on the living subject. An abscess about the size of a pigeon's egg was found in the white matter of the brain, to the inner side of the third left frontal convolution.

In August 1888 [5] Macewen reported his pioneer work on the surgery of the brain and the spinal cord. He described 21 operations performed on patients for cerebral abscess, with 18 recoveries. Years later Dr. Charles Frazier, of Philadelphia, wrote: "Macewen's mortality rate in the series of operations for brain abscess compares more than favorably with the best today."

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2. Guthrie, cited by Wilson.[1]

3. Bowman, A. K.: The Life and Teaching of Sir William Macewen: A Chapter in the History of Surgery, London, William Hodge & Co., Ltd., 1942.

4. Ballance, C., cited by Bowman.[3]

5. Macewen, W.: Address on the Surgery of the Brain and Spinal Cord, Brit. M. J. **2**:302-309, 1888.

Macewen was the originator of aseptic surgery, a development from Lister's antiseptic surgery. His biographer states:

> . . . he was first in the successful practice of laryngeal intubation; first in bone-grafting; first to define cerebrospinal surgery; . . . first to describe precisely the differential processes of wound healing. He was influential in the development of surgery of the mastoid. In addition he was an investigator of a host of activities which covered the whole field of biology.

In preparation for his description of the surgical importance of the "Macewen triangle," he inspected 450 bones, finding the triangle in 426; in 22 of the remaining 24 it was recognizable. It was therefore present in 99.5 per cent of all the specimens, and well marked in 94.6 per cent.

ADVANCES IN THE ANATOMY AND THE PHYSIOLOGY OF THE AUDITORY AND OLFACTORY CENTRAL MECHANISMS

Olfactory Bulb as Observed in Various Vertebrates.—Crosby and Humphrey [6] studied the nuclear configuration of the olfactory and accessory olfactory formations and the nucleus olfactorius anterior in different vertebrates. Serial sections were made from six species of turtles, four of snakes, one of crocodiles, five of lizards, six of birds and ten of mammals. The olfactory bulb showed a wide range in size. In general, this was in direct proportion to the degree of development of the bulbar centers and in inverse proportion to the amount of forward specialization of the hemisphere.

Origin and Abnormalities of the Internal Auditory Artery.—In 70 per cent of the cadavers examined by Brunetti and Caramagna [7] the internal auditory artery sprang from the anterior inferior cerebellar artery (Stopford, 1916). Very rarely it sprang from the basilar artery. Occasionally it was made up of two or three trunks.

Number of Fibers in the Acoustic and Vestibular Nerves.—Rasmussen [8] counted the nerve fibers in 37 vestibular nerves and in 40 cochlear nerves from persons with normal hearing. Apparently, normal cochlear nerves contained from 22,800 to 40,000 nerve fibers; the average in man was 31,400, compared with nearly 50,000 in the cat. The number of vestibular fibers varied from 14,200 to 24,000, averaging 18,500. The

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6. Crosby, E. C., and Humphrey, T.: Studies of the Vertebrate Telencephalon: The Nuclear Configuration of the Olfactory and Accessory Olfactory Formations and of the Nucleus Olfactorius Anterior of Certain Reptiles, Birds and Mammals, J. Comp. Neurol. **71**:121-213 (Aug.) 1939; abstracted, Arch. Neurol. & Psychiat. **44**:187 (July) 1940.

7. Brunetti, F., Jr., and Caramagna, V.: Origine e compartamento della arteria uditiva interna, Valsalva **14**:413-428 (Sept.) 1938.

8. Rasmussen, A. T.: Studies of the Eighth Nerve of Man, Laryngoscope **50**:67-83 (Jan.) 1940; abstracted, Arch. Otolaryng. **33**:1079 (June) 1940.

cochlear nerves of the older subjects contained 2,200 fewer fibers, and the vestibular nerves, 1,000 fewer fibers, than those of the younger subjects.

Rasmussen found that the funicular (cordlike) pattern of the eighth nerve varies greatly in different persons and in different regions of the same nerve.

Technic.—Because of the close association of the cochlear and the vestibular fibers throughout most of their course, the number of cochlear fibers was determined by counting the nerve fibers in the intrapontile portion of the vestibular nerve, the resulting figure being deduced from the total number of fibers in the extramedullary portion of the whole eighth nerve.

The Complex Nature of the Primary Auditory Nuclei and the Bilateral Representation of Each Cochlea.—The organization of the cochlear nuclei appears to be even more complicated than that of either the retina or the olfactory bulb; they function as the primary nuclei of the highest type of neural differentiation in the nervous system (Cajal 1933). Kemp, Coppee and Robinson [9] have stated that no less than forty or fifty types of neurons are present in the primary cochlear nuclei and that each cochlear fiber establishes connections with many hundreds, perhaps thousands, of cells. Each primary cochlear fiber has connections with numerous cells in no less than thirteen regions in which the neurons are found to possess different types of specific structure. The bilateral representation of each cochlea seems to be in the lateral lemniscus of the pons.

"Cochlear Electrical Response" After Section of the Eighth Nerve.—The eighth nerves of 8 cats were cut subdurally and then studied histologically by Guttman and Barrera.[10] The "cochlear electrical response" still persisted two to eighteen months postoperatively. After the animals were killed, histologic studies demonstrated degeneration of the cochlear ganglion and nerve. Thus, the cochlear potential does not depend on the action current of the eighth nerve. On the other hand, the action current of the eighth nerve is dependent on the integrity of the cochlear potential.

Cerebral Acoustic Area in Animals.—The cytoarchitectonic boundaries of the brain's area giving responses to auditory stimuli as determined by Kornmüller [11] include (*a*) the upper part of the sylvian gyrus,

9. Kemp, E. H.; Coppee, G. E., and Robinson, E. H.: Electric Responses of the Brain Stem on Unilateral Auditory Stimulation, Am. J. Physiol. **120**:304-322 (Oct.) 1937. Krieg, W. J. S.: Functional Neuroanatomy, Philadelphia, The Blakiston Company, 1942, p. 144, fig. 97.

10. Guttman, J., and Barrera, S. E.: The Electrical Potentials of the Cochlea and Auditory Nerve in Relation to Hearing, Am. J. Physiol. **120**:666-670 (Nov.) 1937.

11. Kornmüller, A. E.: Die bioelektrischen Erscheinungen der Hirnrindenfelder mit allgemeineren Ergebnissen zur Physiologie und Pathophysiologie des zentralnervösen Griseum, Leipzig, Georg Thieme, 1937.

(*b*) the posterior part of the anterior ectosylvian gyrus and (*c*) the middle ectosylvian gyrus. "Ecto" in "ectosylvian" is from the Greek ektos, meaning outside. Bremer and Dow[12] think that this region represents the auditory projection area in the cat.[13]

> The cerebral acoustic area . . . is identical with the area of projection of the fibers from the medial geniculate body as recently determined in two cats by Wolliard and Harpman (1939).
>
> In confirming the boundaries of this area we do not wish to infer that it is necessarily strictly uniform in cytoarchitectonic structure. Indeed, more rigid criteria than we have been able to apply to this limited material will possibly show the existence of subdivisions within the acoustic area thus eliminated.

Cerebral Acoustic Area of the Monkey as Localized by the Oscillograph.—In the monkey the maximum cortical responses of the acoustic area occurred in the angle formed by the junction of the posterior and medial surfaces of the superior temporal gyrus (Ades and Felder[14]). The stimulus was a sharp click recorded oscillographically.

Tuning Forks in the Measurement of Vibration Sense.—Vibration sense belongs to the group of proprioceptive sensations, including those of position, deep pressure, weight, form and vibration. (Roth[15]). A means for measuring vibration sense is the neurometer, which is a tuning fork especially designed, constructed and laboratory tested as to frequency (128 cycles); intensity (70 decibels at the maximum) and perception time for normal subjects (thirty-five seconds at the fingers). Young persons have a range between 35-35 and 40-35 for normal. Middle-aged persons have a range between 30-35 and 35-35. A 15 per cent margin includes the expected variations of acuity for age.

Vertigo, Nystagmus, Lateral Propulsion (Toward the Side of the Lesions) and Paralysis of the Throat Due to Thrombosis or Embolism of the Posterior Inferior Cerebellar Artery.—The syndrome of the posterior inferior cerebellar artery is caused by an involvement of the dorsal and ventral spinocerebellar tracts. The symptoms are (*a*) ipsilateral lateropulsion (the patient has a feeling of falling to the side of the lesion), (*b*) hemiasynergia, lack of cooperation or of working together of the muscles of one side (this is shown by the patient's past pointing in the direction of the lesion) and (*c*) hemiataxia (the patient presenting adiadokokinesis and tremor).

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12. Bremer, F., and Dow, R. S.: The Cerebral Acoustic Area of the Cat, J. Neurophysiol. **2**:308-318 (July) 1939.

13. Poljak, S.: The Connections of the Acoustic Nerve, J. Anat. **60**:465-469, 1926.

14. Ades, H. W., and Felder, R.: The Acoustic Area of the Monkey (Macaca Mulatta), J. Neurophysiol. **5**:49-54 (Jan.) 1942; abstracted, Arch. Neurol. & Psychiat. **48**:1000 (Dec.) 1942.

15. Roth, A.: Measurement of Vibration Sense, J. M. Soc. Cape May County, New Jersey **5**:3 (Nov.) 1943.

Wallenberg [16] was the first to give a detailed description of the symptoms of the syndrome of the posterior inferior cerebellar artery (1895). So the syndrome is sometimes known as the "Wallenberg syndrome."

The posterior inferior cerebellar artery (the longest branch of the vertebral artery), as pointed out by Cinelli,[17] winds backward around the olive and ascends behind the roots of the glossopharyngeal and vagus nerves to the vallecula of the cerebellum (the deep hollow on the inferior surface of the cerebellum, between the hemispheres, where the medulla oblongata rests).

The main trunk of the artery supplies end arteries to the medulla oblongata and the dentate nucleus of the cerebellum, which are of fundamental importance in the production of the syndrome because they are end arteries.

Occlusion of the posterior inferior cerebellar artery completely shuts off the blood supply of the structures in the dorsolateral aspect of the medulla but only incompletely interferes with the blood supply of the cerebellum, with the exception of the dentate nucleus.

(*a*) Cause of the Vertigo: The syndrome of the posterior inferior cerebellar artery is usually ushered in with vertigo due to the sudden irritation of the triangular and vestibular nuclei. The dizziness soon subsides because there is a compensatory reaction from the central nucleus on the opposite side.

(*b*) Central Nystagmus: The central nystagmus is constant as long as the irritation of the nucleus is present and, after a time, is not associated with dizziness. The slow component of the nystagmus is directed toward the lesion.

Caloric irrigation of the ear on the side of the lesion will always produce normal peripheral nystagmus, with normal concomitant dizziness and past pointing.

(*c*) Ipsilateral Paralysis of the Soft Palate and the Vocal Cord: The ipsilateral paralysis of the soft palate and the vocal cord and the difficulty in swallowing (dysphagia) are due to the involvement of the nucleus ambiguus, which is a long column of nerve cells giving origin to motor fibers that run by way of the glossopharyngeal, vagus and accessory nerves to supply the musculature of the pharynx and the larynx.

Cinelli's Case.—In the case reported by Cinelli,[17] the central nystagmus, which was constant for forty-five days, is accounted for by the irritation of one of the vestibular nuclei, namely, the nucleus triangularis. The left vocal cord was completely paralyzed (accounting for the patient's hoarseness); the left side of the

16. Wallenberg, A.: Acute Bulbar Affection, Arch. f. Psychiat. **27**:504, 1895.

17. Cinelli, A. A.: Syndrome of the Posterior Inferior Cerebellar Artery, Arch. Otolaryng. **36**:108-119 (July) 1942; abstracted, Quart. Rev. Otolaryng. **1**:350 (Sept.) 1942.

soft palate was also paralyzed. There was apparently "complete loss of sensation" in the soft palate and the pharynx, which is explained by the involvement of the sensory fibres of the glossopharyngeal nerve.

Advances in Cerebral Localization of Hearing (Nielsen[18]).—(*a*) Physiologic Aspects of Lesions Causing Auditory Agnosia: A lesion in the "area of Wernicke" causes auditory agnosia. The printed image is tied up with the sound of the word. Before the printed word is called to the attention of the child it has understood the auditory word for four or five years. The engrams[19] of the auditory sounds are more deeply ingrained and function with much greater ease, because spoken language continues to be used throughout life far more than the written word.

The removal of the sound image of the word, therefore, in most cases removes so important a portion of the word (as it exists in the cerebral engrams) that recognition of the word is lost. This is the explanation of the appearance of "Wernicke's aphasia" (which includes loss of recognition of the word seen) from a lesion of the limited area called Wernicke's area. The small portion of the superior temporal convolution between Wernicke's area and the angular gyrus assumes enormous importance in the light of recent investigation. Destruction of this small area (the posterior part of area 22 of Brodmann) has the same effect on reading as does, in most persons, a lesion of the angular gyrus, because the lesion separates Wernicke's area from the angular gyrus.

Nielsen states: Area 19 of Brodmann is certainly divided physiologically; areas 41 and 42, which serve the function of cortical reception of auditory impulses in man, are not present in certain primates, although those primates hear apparently as well as man.

The function of the angular gyrus is somewhat different in different persons so far as recognition of symbols is concerned. This difference depends on education and training. The general rule is that the angular gyrus can, without help from the motor area, serve for recognition but that it needs association with the auditory area certainly for interpretation and also, in many cases, for recognition of written symbols. This is due to the fact that when a person is taught to read, the auditory memories of the spoken words are always brought into play.[20]

In the sphere of hearing the engrams which constitute the anatomic basis of memory are located not in the primary area for perception

18. Nielsen, J. M.: A Textbook of Clinical Neurology, New York, Paul B. Hoeber, Inc., 1940, pp. 233, 266 and 268.

19. In the act of observation one normally forms engrams, or neurograms, which are the anatomic basis of memory. An engram is not a new anatomic structure but a previously existing set of cells, axons and dendrites trained by practice to form a "beaten pathway" over which impulses travel with greater ease than over untrained pathways. As they are in the nervous system they are sometimes called neurograms.

20. Nielsen,[18] pp. 229 and 237.

but in the secondary area (Wernicke's area) of each side. Destruction of the subcortical fibers does not wipe out memories. There was the deaf Beethoven, who could reauditorize sound perfectly. He could even auditorize written music which he had never heard. This fact illustrates that Wernicke's area for reauditorization can be aroused from any part of the cortex.

(*b*) Experimental and Operative Removal of Hearing Centers: In case of destruction of Wernicke's area of the major side, the patient loses his power of identifying spoken language. Sounds in general are usually still recognized by the other (minor) side, for which reason loss of comprehension of sounds in general is rare.

In case of destruction of the anterior half of the major superior temporal convolution, recognition of music is temporarily abolished. Henschen has shown that unilateral dominance is slight in the auditory sphere of music, and in the great majority of cases the minor (usually the right) side easily takes up the function. Nielsen states:

> The entire temporal lobe has been removed surgically in a number of cases, after which hearing in general is slightly reduced in the opposite ear. Yet this reduction is hardly noticeable. The anatomic basis for this fact is that both of the mesial geniculate bodies receive fibers from each cochlea.

However, bilateral lesions of the mesial geniculate bodies or of the auditory radiation do produce total deafness. The auditory radiations go to the superior border of the temporal lobe, where they are hardly ever involved by the usual lesions of this area, such as tumor or abscess of the temporal lobe, or by softening in the distribution of the middle cerebral artery.

(*c*) Otitic Abscess of the Temporal Lobe Often Causes Hemianopsia: Fibers convey impulses from the ipsilateral sides of both retinas, including the ipsilateral halves of the maculas. However, while the fibers go directly to the calcarine area, they do not go straight. The lower ones sweep forward into the temporal lobe and spread out in a curve, going forward nearly as far as the optic chiasm. This loop (Flechsig's) has been described by Adolph Meyer and is known as Meyer's loop, but some call it Cushing's loop.

The fibers of Flechsig's (Meyer's) loop, which sweep into the temporal lobe before going backward lateral to the posterior horn of the lateral ventricle, are subject to disease affecting the temporal as well as the temporo-occipital lobe. An otogenous temporal abscess located in the usual site just above the auditory meatus may compress the loop or cause its functional deletion by inflammatory reaction.[21]

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21. Eagleton, W. P.: Brain Abscess, New York, The Macmillan Company, 1922. A transient suggestive hemianoptic field in a case of abscess of the adjacent left temporosphenoidal lobe is illustrated in figure 37 on page 180.

(*d*) Auditory Hallucinations: Stimulation of the auditory fibers or of the auditory cortex as by a vascular lesion, an abscess or a tumor will give symptoms — hallucinations. Courville studied patients with temporal tumor from Cushing's clinic and found auditory hallucinations much more common than is usually supposed. Hallucinations do not depend on laterality of the lesion. They are a not infrequent result of tumor or abscess of either temporal lobe. The hearing of voices is less common than the hearing of music. If the sounds are due to temporal trouble they are associated with a belief of reality and are not recognized as mere "hissing in the ear" (Nielsen).

The final step in the establishment of Wernicke's area as the center for recognition and reauditorization of sounds came from Foerster, who actually succeeded in producing auditory hallucinations of spoken language by electrical stimulation of the area outlined.[22]

ACCIDENTS IN AURAL AND NASAL SURGERY

Air Embolism.—McGovern [23] reports 2 cases of air embolism following antral puncture, one of which was fatal. He advises (1) extreme caution in the avoidance of trauma and (2) in the use of low pressure.

(*a*) Symptons of Cerebral Air Embolism: The onset is abrupt, occurring immediately after the insufflation of air. The patient may complain of local pain and exhibit great anxiety and restlessness. Sudden pallor is followed by cyanosis, convulsive twitching or generalized convulsions, irregular pulse, loss of consciousness and irregular stertorous respirations. Death from respiratory paralysis or cardiac standstill results from coronary embolism or cerebral embolism following the entrance of air under high pressure with open vessels.

(*b*) Fatal Case of Air Embolism Following Air Inflation of a Maxillary Sinus: A diagnostic puncture of the right maxillary sinus of a 24 year old man was made by way of the inferior meatus. After the insufflation of air and the withdrawal of the needle the patient had a severe convulsion. His breathing became stertorous and soon ceased. Prolonged artificial respiration and administration of stimulants failed to revive him.

(*c*) Recovery from Air Embolism Following Air Inflation of a Maxillary Antrum in a Diabetic Patient: A 24 year old woman had been restricted to a diabetic diet. She had right-sided acute maxillary sinusitis of three weeks' duration. Pus was removed by irrigating the antrum by means of a cannula inserted through the natural ostium, after which the antrum was treated by insufflation of air.

Immediately on withdrawal of the cannula the patient had a generalized convulsion, with stertorous breathing. On recovery from the convulsion she remained in partial coma, with extreme restlessness, hyperactivity of the knee reflexes and slight weakness of the left arm and hand.

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22. Nielsen,[18] pp. 231, 240-242 and 246.

23. McGovern, F. H.; Cerebral Air Embolism Following Antral Irrigation, Arch. Otolaryng. **34**:593-595 (Sept.) 1941.

Although the blood sugar was 312 mg. per hundred cubic centimeters, the lack of acetone bodies in the urine and the absence of the usual picture of diabetic coma suggested the diagnosis of "cerebral air embolism similar to that following artificial pneumothorax."

On the following day the weakness became more pronounced, but the condition improved the next day. Within a week, recovery was complete.

(*d*) Air Embolism Following Operations on the Neck: Guthrie [24] stated that many unexplained deaths result from air embolism, especially in operations on the neck. He reported 3 cases of serious and 1 case of fatal air embolism occurring during thyroidectomy.

(*e*) Air Embolism During Incision of the Lateral Sinus: This procedure is rarely followed by air embolism.

(*f*) Air Embolism Following Eustachian Catheterization: Blegvad [25] encountered 4 cases of air embolism following air inflation of the middle ear. One was fatal. Ramberg [26] reported a case in which air embolism followed inflation of the eustachian tube; there was a sudden apoplectiform seizure, in which apparently both cerebral and cardiac embolisms occurred. The seizure was accompanied by conjugate deviation of the eyes to the left, snoring respirations, dilation of the pupils, which did not react to light, red cyanosis followed by pale cyanosis, cessation of respiration and cardiac standstill. Artificial respirations restored breathing, and consciousness was regained.[27]

Fatal Intracranial Venous Obstruction Following Nasal Displacement Irrigation in the Presence of Vascular Degeneration in a Young Person.—Furrer, Stanard and Kuykendall [28] report this case:

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24. Guthrie, D.: Die Gefahren der Luftembolie mit besonderer Berücksichtigung der Thyreoidektomie, Wien. klin. Wchnschr. **50**:1667-1670 (Dec. 10) 1937; abstracted, J. A. M. A. **110**:541 (Feb. 12) 1938.

25. Blegvad, N. R.: Luftembolie bei Luftdusche, Ztschr. f. Hals-, Nasen- u. Ohrenh. **45**:363-378, 1940.

26. Ramberg, J.: Some of the Risks Associated with Catheterization of the Eustachian Tube, Acta oto-laryng. **28**:482-492, 1940.

27. The literature on air embolism in general includes the following articles: McDaniel, S.: Air Embolism: Causę and Treatment, Dis. of Chest **7**:146-149 (April) 1941. Bierhaus, H., and Hintze, H. E.: Ueber die Todesursache bei venöser Luftembolie, Arch. f. klin. Chir. **201**:1-26, 1941. von Balogh, E.: Bausteine zur lebensrettenden Therapie der venösen Luftembolie, Virchows Arch. f. path. Anat. **307**:362-369, 1941. Osborn, G. R., and Dawson, J. C. C.: Air Embolism, with Reports of Three Cases, Lancet **2**:770-772 (Oct. 1) 1938. Hartley, G. S., and Yorkoff, F. H.: Air Embolism or Pleural Shock: Report of Two Cases, Virginia M. Monthly **65**:234-236 (April) 1938. Dible, J. H.; Hewer, T. F.; Ross, A. O. F., and Walsh, C. H.: Air Embolism in Urethroscopy and Tubal Insufflation, Lancet **1**:313-315 (Feb. 5) 1938.

28. Furrer, E. D.; Stanard, D. C., and Kuykendall, J.: Hemorrhage into the Paranasal Sinuses Following Suction Displacement Irrigation, Arch. Otolaryng. **33**:305-307 (Feb.) 1941.

A 23 year old woman with chronic postnasal discharge was relieved by displacement irrigation, 8 cc. of Locke's solution containing an ephedrine salt in 0.5 per cent concentration being used, with a suction pressure of 17 to 20 mm. of mercury. Treatments were given at intervals over several months. Ten months later she complained of pain over the left eye. Three days after this she had numbness in the right hand, which subsided the next day. Later the same day flaccid paralysis of the right arm and forearm developed, and two hours later, paralysis of the face, the right hand and the left leg. She became unconscious. The next day she had a temperature of 102 F. The spinal fluid pressure was 44 mm. of water; there were no cells. The following day opisthotonos and convulsions were followed by cyanosis and death.

Autopsy showed softening of the left parietotemporal lobe and an area undergoing encephalomalacia. The venous sinuses about the pacchionian bodies on the left side contained adherent blood clots. The right sphenoid and ethmoid sinuses were filled with clotted blood. Microscopic sections through the areas of infarction showed necrotic tissue.

The authors express the belief that the changes in the brain were due to thrombophlebitis of veins of the dura resulting from absorption of infective material of the sinuses and that the process antedated the last two treatments. "It is conceivable that the cerebral changes were beginning at the time of the treatment, as the patient complained of pain in the right eye." On the other hand, they conclude that the blood in the sinuses was there as a result of the displacement irrigation.

RESULTS OF NASAL OPERATIONS IN THE TREATMENT OF OPTIC NEURITIS CAUSED BY DISSEMINATED SCLEROSIS

Otolaryngologists report cases of optic neuritis and retrobulbar optic neuritis in which cure has been effected by surgical treatment of infected nasal sinuses, particularly the sphenoid sinus and the posterior ethmoid cells. McIntyre and McIntyre [29] have never observed a case of optic neuritis which was due to an infection of a nasal sinus.

Twenty-seven of the McIntyres' 55 patients with multiple sclerosis had optic neuritis. In only a single patient, however, were both optic nerves affected at the same time. Twenty-four patients with optic neuritis recovered without operation on the sinuses.

Two of their patients were seen first by otolaryngologists, who performed operations on the ethmoid sinuses. The vision of both these patients improved. One patient had palsy of the sixth and seventh nerves during his first attack of multiple sclerosis.

Comment (W. P. E.).—No vestibular tests were made in any of the cases of multiple sclerosis. Optic neuritis due to multiple sclerosis is differentiated from that due to inflammatory disease of a sinus by: (1) absence of tenderness on pressure of the eyeball, for in inflamma-

29. McIntyre, H. D., and McIntyre, A. P.: Prognosis of Multiple Sclerosis, Arch. Neurol. & Psychiat. **50**:431-438 (Oct.) 1943.

tory disease of a sinus the zonula of Zinn, which surrounds the optic foramen,[30] causes pain on pressure in the inflammatory process, and (2) a greatly increased duration of the nystagmus following turning which is absent in sinus disease.

DEAFNESS AND DISTURBANCES OF HEARING IN GENERAL DISEASES

Psychic Deafness in Children, Better Named Cerebral Deafness.—(*a*) Psychology of Hearing: Hearing consists of two major parts—(1) the end cerebral organ and reception and perception of the sound waves, and (2) listening. Together they make up hearing.[31]

Froeschels[32] states that experts agree that in some persons hard of hearing from an organic lesion, hearing is improved by psychologically "awakening their attentiveness to acoustic stimuli"; in other words, by "educating the centers to function up to their highest capacity."

(*b*) Hearing Centers Stimuli Absorb Like a Sponge During Infancy: Normally developed centers in infants receive stimuli from the outer world much as a sponge absorbs water. For no active collaboration of the will can be assumed at the earliest period of life. Thus the cerebral centers "become charged" in a purely passive way. When the first mental activities are demanded—for example, when the infant is called on to repeat words—the demand finds the mind prepared by the impressions which it has passively acquired.

If the cerebral center or centers are not normal, what is asked of the child will be more than he can do.

Froeschels thinks that in the condition called "hysterical deafness of childhood" there is in reality a slight anatomic or physiologic defect of the transverse temporal gyrus of Heschl. This anatomic or physiologic defect produces a "disproportion" between the child's (1) "inner readiness" and (2) the demands produced on the child's mind by the outside, which leads to a "feeling of uneasiness on the part of the child." It is this disproportion between (1) inner readiness and (2) demand from the outside that leads to a feeling of mental uneasiness in the child. The "psychic component . . . seems to be the natural consequence of the primary defect." Froeschels suggests that the term "central

30. Eagleton, W. P.: Localizing Value of Ophthalmic Examinations in Suppurative Diseases of the Brain: Swelling of the Nerve Head from Suppuration Around the Optic Foramen, J. A. M. A. **92**:713-720 (March 2) 1929; Traumatic and Infective Lesions of the Head, the Chief Manifestations of Which Are Visual Disturbances, S. Clin. North America **6**:1435-1452 (Dec.) 1926.

31. In some children with so-called hysterical deafness, Froeschels found the listening was at fault. W. P. E.

32. Froeschels, E.: Psychic Deafness in Children, Arch. Neurol. & Psychiat. **51**:544-549 (June) 1944.

deafness" more adequately expresses the basic trouble than "psychic deafness."

(*c*) Infrequency of Psychic Deafness in Children: Froeschels has encountered but 12 psychically deaf children among over 60,000 persons with disturbances of speech and voice.

Froeschels reports the case of a 10 year old girl, daughter of congenitally deaf parents, who was thought to be deaf. Her hearing proved to be normal, to her own surprise, when tested with the Urbantschitsch whistles. Stein [33] reports a similar case.

Froeschels' patient at times showed psychic resistance to acoustic stimuli, "an adversion to hearing." Froeschels thinks (1) that inactivity of Heschl's convolution may produce an aversion to hearing and (2) that inactivity of the normal Heschl center may create the picture of deafness.

Cochlear Neuroepithelial Degeneration, the Result of Syphilitic Glioma and of Vascular Infiltration Involving the Medulla Oblongata.—Tamari [34] in summing up his microscopic observations in such a case says: "Visualizing . . . the changes in the cochlea in connection with the [syphilitic] process in the medulla oblongata . . . one has a picture of a protracted, descending, degenerative process of the cochlea in which the epithelial sensory cells deteriorated as a result of the destruction of the . . . fibers" of the auditory nerve.

Tamari's [34] patient, a man of 52, gave a history of an old mercury-treated syphilitic infection. For a year prior to admission there had been attacks of dizziness, tinnitus and impairment of hearing in both ears; recently there had been deafness and for three weeks progressive hoarseness and difficulty in swallowing.

He complained of intense dizziness, with a tendency to fall backward. There was complete deafness. The right pupil was smaller than the left, with sluggish reaction to light. There was horizontal nystagmus to both sides. Sensibility of the face was decreased, particularly on the right side. The tongue was immobile. Swallowing was impossible. The soft palate was immobile. There was decreased sensitivity to pain on both sides of the entire body. The patellar and achilles tendon reflexes were diminished. Death was caused by aspiration pneumonia.

"The histologic pictures of the two cochleas were practically the same." In the internal acoustic meatus the greater part of the space was nearly empty, being only partly filled with nerve fibers. "Here and there lay dispersed isolated pieces of destroyed nerve tissue. The periosteum of the meatus was lifted in places," "and in one place embedded amyloid bodies were seen." The spiral ligament was reduced in size in all the coils. Corti's organ was largely degenerated.

Serial sections of the medulla oblongata showed the following changes: In sections at the level of the beginning of the decussation of the lemnisci there was "dense infiltration in Goll's nucleus and less in Burdach's, particularly in the area of the spinal trigeminal root and also around the central canal. There was perivascular infiltration ventrally on one side and, to a lesser degree, on the opposite side."

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33. Stein, L.: Speech and Voice, London, Methuen & Co., Ltd., 1942, p. 98.

34. Tamari, M.: Atrophic Degeneration of the Cochlea in Syphilitic Gumma of the Medulla Oblongata, Arch. Otolaryng. **34**:1014-1022 (Nov.) 1941.

"The tumor was situated on the roots where the beginning of the cochlear nerve and the area of the lateral recess respectively were distinct and heavily infiltrated. The entrance of the cochlear nerve, the ventral cochlear nucleus, the lateral cochlear root and the tuberculum appeared infiltrated."

The gumma was shown to extend on both sides, into the pons and into the lateral sections of the crura pedunculi. . . . "the facial nucleus was entirely affected. The gumma projected also medially above the superior olive. At this point, both facial nerves were in the gumma, and their medullary sheaths lost their stain as they left the brain."

Summary of Microscopic Observations on the Medulla Oblongata.—The case presented "gummatous infiltration of the medulla oblongata from the beginning of the decussation lemniscorum until about the end of the fourth ventricle in the region of the trochlear decussation. The process ran mainly in the dorsolateral boundaries, involving . . . the nucleus of Deiters in the cerebellar region and the root and nuclei of the cochlear nerve and finally becoming a gummatous tumor in the nuclear area of the fifth and seventh nerves and ending again abruptly as infiltration above this region.

". . . On the opposite side . . . the gumma . . . involved all lateral and dorsal efferent nerves and their nuclei, . . . Further, all lateral and dorsal situated systems, i. e., the nucleus of Deiters, the restiform body, the spinal trigeminal roots, part of the glossopharyngeal root, the olivocerebellar fibers and the outer arcuate fibers, appeared destroyed. . . ."

Alexander's Classification of Syphilitic Processes Which Affect the Auditory and Vestibular Mechanisms.—According to Tamari, Alexander [35] primarily divided syphilitic changes of the ear into (*a*) syphilitic neuro-otits interna, or (*b*) a form conditioned by endarteritis syphilitica, (*c*) a toxic form, and (*d*) a gummatous form. Other authors differentiate syphilitic changes in the membranes of the labyrinth and the nerve apparatus itself. Syphilitic changes in the labyrinthine fluid may result in degeneration of the neuroepithelium, in contrast to a syphilitic attack on the nerve itself. The latter may result in degenerative changes of the nerve stem or in peripheral cochlear degeneration, including all three components of the nerve apparatus, viz., ganglion, nerve fibers and hair cells.

Deafness in Sickle Cell Thrombotic Anemia.—Connell [36] reports a case:

A Negro woman aged 20 was admitted to a hospital, complaining chiefly of headache, right-sided deafness and drowsiness. The headache and the deafness had appeared six days previously; the drowsiness, a few hours later. The headache was limited to the left side. She said that six years previously she had an attack of rheumatism accompanied by left-sided paralysis. Recovery had been complete except for residual weakness of the left arm and leg.

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35. Alexander, G.: Die Syphilis des Gehörorgans, Vienna, A. Hölder, 1915; New Histological Findings in the Ear in Lues and Their Importance in General Pathology of the Ear, Laryngoscope **38**:295-305 (May) 1928.

36. Connell, J. H.: Cerebral Necrosis in Sickle Cell Disease, J. A. M. A. **118**:893-895 (March 14) 1942.

Three days after admission she suddenly became comatose. The pupils did not react to light the left pupil being larger than the right. The neck was stiff, and there was pharyngeal paralysis. No abdominal reflexes could be elicited. Her temperature was 104.6 F. The spinal fluid cell count was less than 10; the pressure was slightly increased.

At postmortem examination the spleen, a miniature organ, weighed only 2 Gm.; the normal tissue was replaced by pigmented fibrous tissue. Its appearance suggested sickle cell disease. Blood smears revealed a severe degree of every type of sickling.

The brain was edematous. The dura was thickened and pigmented throughout, the pigmentation being especially noticeable in the midbrain. There was a firm red blood clot in the left middle cerebral artery. Gross sections of the brain showed (1) massive necrosis of the left cerebral hemisphere, with attending inflammatory reaction, involving chiefly the white matter though sparing the corpus callosum and other major nerve tracts, (2) an old cystic pigmented lesion in the right cerebral hemisphere, and (3) an old cystic pigmented lesion in the right cerebellar lobe.

Microscopic examination of the brain tissue revealed endothelial proliferation and hyalinization of the smaller arterioles. The neurogenic macrophages contained pigment. All the vessels in the brain were filled with greatly distorted sickled red blood cells. The left middle cerebral artery showed a red thrombus with beginning organization.

Sicklemia, according to Connell, is abnormal erythropoiesis. It is a tendency toward sickle formation of red blood cells; that is, it is the production of elongated semilunar erythrocytes, which are destroyed by the body defenses as abnormal cells.

The disease is inherited as a dominant mendelian characteristic and is seen chiefly in the pure Mediterranean races and in the American Negro; the tendency is reported to exist in 7.5 per cent of all members of the Negro race.

The elliptic blood cells, because of their shape, are poorly adapted to pass through the circulation. As a result, they stagnate in the blood stream, causing obstruction, and a state of local anoxemia develops. The smaller arterioles become occluded; the resulting thrombosis terminates in local ischemia and necrosis, especially in those areas in which there is inadequate collateral circulation. Thrombotic lesions of the bone marrow may cause fat embolism (Vance and Fisher).

Summarizing, Connell states: " . . . the fundamental pathologic physiology of sickle cell disease . . . is not anemia . . . but thrombosis. It may therefore involve any organ, particularly any organ with a terminal circulation."

Comment (W. P. E.).—The incomplete and contradictory otologic statements detract from the value of the case. Probably a thrombus in the labyrinthine vessels caused the deafness, as they are terminal arteries with no collateral circulation. From the postmortem observations it would appear that the right-sided deafness and left hemiplegia

were due to a pigmented vascular obliterative lesion of the midbrain, for in another statement the deafness was recent.

Herpes Zoster Oticus: Asherson's Suggestion of a Double Cochlear Nerve.—Asherson [37] thinks that herpes zoster oticus, or porus acusticus polyneuritis, is caused by vascular occlusion. He suggests that as nerve deafness manifests itself by a loss of perception of high tones the supposition is justified that the cochlear nerve is a composite nerve, consisting of at least two main nerves: one part concerned with the transmission of high tones; the other part, with the transmission of low tones.

The nerve trunk transmitting the higher tones would appear to be the more vulnerable of the two trunks. Asherson believes that a lesion involving the branches of the cochlear nerve is always secondary to vascular occlusion. He offers two suggestions: 1. Any noise above a certain intensity is a stimulus which reacts through the ear and produces a spasm of an end artery distributed to that part of the high tone division of the cochlear nerve which transmits the frequency 4096. 2. The fact that the low tone nerve is affected only after the high tone nerve suggests that the end artery branch to this comes off the main trunk more proximally, and possibly has a wider bore.

The apparent vulnerability of the branch transmitting the high tones is due to the fact that the most terminal ramification of the end artery to the cochlear nerve supplies this branch, ending in a minute vessel supplying the part concerned with frequency 4096. Occlusion of this (arterial disease) paralyzes the high tone branch, and "high tone deafness" results. If the occlusion spreads more proximally, the vessel to the low tone division is also involved, and both high and low tones fail to be perceived at normal levels. The perception of low tones is never affected in cases of "nerve deafness." The condition would probably be mistaken for deafness due to catarrh of the middle ear. Early restoration of the blood supply, whether by recanalization of the occluded vessel or by establishment of collateral circulation, will lead to early recovery of the nerve function.

Asherson enumerates the following causes of occlusion: The vessel may be closed by (*a*) spasm (then deafness will be transient), (*b*) embolism (secondary to sepsis elsewhere in the system), (*c*) thrombophlebitis obliterans (infection spreading from a neighboring focus), or (*d*) endarteritis obliterans associated with arteriosclerosis in the elderly (permanent occlusion of the vessel)—hence the frequency of presbyacusis— or (*e*) syphilitic endarteritis obliterans, which is the nerve deafness of syphilis, hereditary or acquired.

37. Asherson, N.: The Cochlear Nerve and the Vascular Theory of Nerve Deafness, J. Laryng. & Otol. **55**:531-545 (Dec.) 1940.

In support of these theoretic suppositions he offers the following clinical observations: 1. The nerve deafness among the aged (referred to as presbyacusis) "manifested itself by a loss of perception of high tones, the low tones being in many cases perceived normally." 2. "The persistent noise of an aeroplane propeller (a *low pitched* sound) will produce a nerve deafness manifested by a loss of *high* tones." 3. Among boiler makers "a low tone noise resulted in loss of high tones—i. e., the accepted nerve deafness" (Larsen 1939).

Asherson's first case is unique in that with acute paralysis of the entire cochlear nerve the patient recovered, and a consecutive series of audiograms of the progress and the recovery is available.

The patient, a young woman, complained of vertigo. In the early stage, before she was aware of any impairment in hearing, the audiometer detected a gap around the frequency 4096.

The sole physical sign noted was loss of the glabella reflex on the right side, the patient blinking only with the left eyelids. This was the first and only physical sign of impending facial paralysis, which developed a few days later. The early detection of the loss of the glabella reflex not only identified the ear which was the source of the vertigo (the right ear) but enabled an exact diagnosis of polyneuritis (herpes zoster oticus type) to be made.

Asherson suggests that, "the paralytic wave" swept across the seventh and the eighth nerve in the following chronologic order, viz.: the vestibular nerve, the facial nerve, the high tone branch of the cochlear nerve and the low tone branch of the cochlear nerve. The facial paralysis soon became well marked and complete. A few days later the paralysis had extended, and the audiogram now showed a loss of the low tones also, the loss being of the same extent as that of the high tones; by now both branches of the cochlear nerve were paralyzed. Conversational voice could be perceived loudly only at 1 foot (about 30 cm.).

The vestibular nerve was also involved, as there was no response to syringing the right ear with water at 70 F.

The face showed signs of improvement while the lesion was extending in the cochlear nerve, and Asherson was able to predict the date at which the hearing would be restored to normal from the time which the facial nerve took to regain function. The order of recovery was in the order of the onset, and this was reflected in the audiograms. The paralytic phenomena subsided within twenty-eight days, by which time the facial paralysis had disappeared completely.

On the fourteenth day the hearing in the affected right ear was suddenly lost. The deafness was profound, and the conversational voice could be perceived at 1 foot only. The audiogram taken at this stage showed a great loss of perception for high tones—frequencies above and including 1024—characteristic of what is usually accepted as "nerve deafness." Within fourteen days after the deafness, though the patient was unaware of any alteration, the audiogram indicated recovery of hearing as regards the high tones, the low tones at this stage being lost to the same extent. By the end of the third week, the low tones had been restored.

This patient had acute vertigo due to commencing polyneuritis of the vestibular and facial nerves and two branches of the cochlear nerve. A series of audiograms showed the changes in the hearing during the period of invasion, when the high tones were affected first and the low tones later, and during the recovery, which took place in the reverse order.

March 19, 1940: Giddiness had been present for ten days. The hearing of the right ear was unaffected. The audiogram confirmed the evidence of normal hearing of the right ear. There was rotatory nystagmus to the left on looking left. There was absence of the glabella reflex. The patient blinked spontaneously only with the left eyelids, the right lids remaining motionless.

March 27: Paralysis of the right side of the face was now complete. There was a salty taste in the roof of the mouth on the right side (involvement of the chorda tympani); there was hyperacusis (stapedial involvement).

April 3: High-pitched tinnitus and deafness had developed in the right ear. There was paralysis of the lower half of the right side of the face. The patient closed the eyes voluntarily. The glabella reflex was absent on the right. Conversational voice was perceived only at 1 foot. Weber's test showed sound lateralized to the left ear, the better one. Bone conduction on the right side was diminished. There was loss for high tones.

April 9: Tinnitus was noted; the audiogram showed marked loss for all tones, high and low, of the same degree as that of the high tones six days previously. A caloric test with water at 70 F. in the right ear produced no response.

There was a sensation of pins and needles in the legs at night, but neurologic examination revealed no abnormal physical signs, the reflexes being normal.

Vitamin B was given by mouth and three intramuscular injections of Berin Forte (thiamine hydrochloride).

Subsequent Progress: Within four weeks the lesion of the facial nerve had subsided, and soon after that the hearing was restored to normal, the patient recovering except for the sensation of pins and needles in the limbs, which took weeks to disappear.

Comment (W. P. E.).—This case of Asherson's undoubtedly is one of the Ramsay Hunt syndrome but is unusual in the absence of herpes in the ear and in the presence of a sensation of pins and needles in the limbs at night. The pathologic aspects have been discussed by Maybaum and Druss [38] and summarized in Eagleton's Index in a case of periarteritis nodosa with hemorrhages in the labyrinth and degeneration of the cells of the left geniculate ganglion.

Asherson's suggestion of the cochlear nerve being formed of two parts, one transmitting high tones and the other low tones, is contrary to present knowledge of the transmission of sound waves through and beyond the labyrinth.

A plausible explanation of the changes in hearing is that a localized inflammatory exudate occurring in the labyrinth itself involved the nerve fibers ending in the lower part of the basal turn of the cochlea, which evoked auditory sensations of high pitch (Stevens and Davis [39]).

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38. Maybaum, J. L., and Druss, J. G.: Geniculate Ganglionitis (Hunt's Syndrome), Arch. Otolaryng. **19**:574-581 (May) 1934; abstracted, Eagleton, W. P.: Eagleton's Index and Abstracts of Literature on Progress in Intracranial Lesions Related to Aural and Nasal Conditions, Newark, N. J., 1941, reprint (1937), pp. 39-40.

39. Stevens, S. S., and Davis, H.: Hearing, New York, John Wiley & Sons, Inc., 1938, p. 352.

Fresh hemorrhagic exudate in the cochlea was found at autopsy by Maybaum and Druss.[38]

Paralysis of the Stapedius Muscle from Herpes Zoster Oticus.—The literature indicates that herpes zoster oticus of wide extent is rare and that the deafness is usually permanent. However, in Stock's case [40] hearing was completely restored. The deafness was diagnosed with tuning forks as nerve deafness. The symptoms of paralysis of the stapedius muscle were unique.

The patient, a woman aged 57, had severe intermittent "shooting" right-sided occipital headache. On the seventh day there was burning pain in the lobule of the right ear, the right half of the tongue and the right half of the soft palate, followed on the eighth day by a herpetic eruption of the right ear beginning on the lobule and extending over the entire lateral surface of the auricle. A herpetic eruption developed over the right half of the soft palate and tongue. On the tenth day the patient had vertigo, nausea and vomiting, nystagmus, and paralysis of the right side of the face. The vertigo was absent as long as she remained on her back but became violent on the least movement of her head.

On the tenth day examination revealed a herpetic eruption of the entire right nasal chamber. There were complete paralysis of the right side of the face and third degree spontaneous rotatory nystagmus, with the fast component to the left. Hearing grossly was normal.

The vertigo abated gradually during the eleventh and twelfth days, and on the thirteenth day the patient was able to be up. On the fourteenth day tinnitus and deafness developed in the right ear. By the nineteenth day the nystagmus and the vertigo were improved. On the twenty-eighth day taste seemed normal. On the thirty-sixth day the patient complained that all sounds caused rattling in the right ear. Hearing had improved; the tinnitus had diminished.

It was felt that the rattling induced in the ear by all perceptible sounds was due to paralysis of the stapedius muscle. The symptom had been rendered obvious by the improvement in hearing.

On the fiftieth day the nystagmus was of the second degree, with the fast component still to the left. All perceptible sounds still caused rattling in the right ear. The spoken voice was heard at 5 feet (1.5 meters) and the whispered voice at 8 inches (about 20 cm.). The tinnitus was diminishing. Tuning fork tests showed nerve deafness.

On the seventy-first day there was no nystagmus, and the spoken voice was heard in the right ear at a distance of 30 feet (9 meters) and a whisper at 10 feet (3 meters). There was no tinnitus, but sounds still caused slight rattling. "Caloric tests produced no reaction in the right semicircular canals and a normal reaction in the left semicircular canals." The facial paralysis was still complete.

The treatment consisted in administration of thiamine hydrochloride and vitamin B complex, with galvanic stimulation of the involved muscle.

Rare Cases of Herpes Zoster Oticus.—In the case of herpes zoster reported by Negus and Crabtree [41] there was no involvement of the

40. Stock, M. F.: Herpes Zoster Oticus, Arch. Otolaryng. **36**:560-561 (Oct.) 1942.

41. Negus, V. E., and Crabtree, N. C.: Case of Herpes Zoster of the VIIth, IXth and Xth Cranial Nerves, J. Laryng. & Otol. **58**:192-195 (May) 1943.

eighth nerve. The course of the disease suggested involvement of the geniculate, glossopharyngeal and vagal ganglions followed by paralysis of the motor fibers passing through them. All the fibers of the seventh nerve were involved as they traveled through the geniculate ganglion.

The involvement of the various branches of the ganglion itself gave rise to localized signs. Thus that of the great superficial petrosal nerve, passing through Meckel's ganglion, led to vesication on the hard palate, supplied by the lesser palatine branches. The same involvement accounted for absence of lacrimation, by virtue of inactivity in the branch of the sphenopalatine ganglion to the lacrimal gland.

A sign of involvement of the glossopharyngeal nerve was anesthesia of half of the pharynx and edema of the faucial pillars and of part of the palate, the posterior margin of which lies in the area of distribution of this nerve; it is probably the petrosal and not the jugular ganglion which is affected by herpes, according to previous reports.

Sherrington described this distribution of the auricular branch of the vagus, arising from the ganglion of the root and usually known as Arnold's nerve, with which a small communicating branch from the geniculate ganglion forms a union.

The pharyngeal branch of the vagus was also involved, with paralysis of constrictor muscles on the right side.

The superior laryngeal branch of the vagus showed its involvement by the presence of herpetic vesicles on the epiglottis and the aryepiglottic fold.

From the pharyngeal plexus, formed by union of the pharyngeal branch of the vagus with the glossopharyngeal nerve, a branch is sent to supply the levator palati muscle, whose lack of action resulted in immobility of half of the palate; the motor fibers arise in the nucleus ambiguus and pass down in the vagus.

The recurrent laryngeal nerve also was affected, with paralysis of the right vocal cord; fibers to the intrinsic laryngeal muscles are derived from the motor nucleus of the spinal accessory nerve, which join the vagus at the ganglion of the trunk (nodosal ganglion); this anatomic fact demonstrated that the lower ganglion and not the ganglion of the root (jugular ganglion) must have been affected, although the latter may also have been involved.

The course of the disease showed a succession of symptoms and signs, herpetic eruption on the pinna being succeeded by dysphagia and dysphonia, and these again by facial paralysis. This would suggest affection of the cells in the geniculate, glossopharyngeal and vagal ganglions, followed by paralysis of motor fibers passing through.

In a case of deafness and loss of vestibular function following herpes of the scalp, Kelly [42] felt that the ganglions involved were the semilunar ganglion of the fifth nerve, the posterior root ganglions of the second and third cervical nerves and the ganglions of the eighth nerve—the spiral ganglion and Scarpa's ganglion.

Pathology of Herpes Zoster.—The disease is probably due to a filtrable virus similar to, if not identical with, that of varicella, which provokes an acute inflammatory reaction in isolated spinal or cranial sensory ganglions, the posterior gray matter of the spinal cord and the adjacent leptomeninges. The clinical manifestations are vesicular cutaneous eruption, radicular neuralgia and, less often, segmental palsies and sensory loss.

Ramsay Hunt described the postmortem changes in a single case. The geniculate ganglion was not examined. Denny-Brown and associates [43] have found only one other histologic study of the "geniculate syndrome" in the literature, that of Maybaum and Druss,[38] who reported a case of periarteritis nodosa in which palsy of the face was present three months before death and pain, with blebs on the tympanic membrane, the auricle and the pinna, six weeks before death. The cells of the geniculate ganglion were shrunken, pyknotic and, in some instances, disintegrated.

Hemorrhagic Lesion with Parinaud's Syndrome from Hemorrhage at the Base of the Third Ventricle Ushered in by Tinnitus.—Parinaud's syndrome consists of paralysis of the rectus lateralis muscle of one eye and spasmodic contraction of the rectus medialis muscle of the other eye.

In van Gehuchten's [44] case, Parinaud's syndrome began with tinnitus.

The patient was a man aged 66. Sudden tinnitus was followed by loss of consciousness and paralysis of all four extremities. The coma persisted for several days. After regaining consciousness the patient had paralysis of the right side of the face and body, pain in the right half of the face, and complete paralysis of the upward gaze.

At autopsy a hemorrhagic area was found in the midbrain at the base of the third ventricle surrounding the aqueduct, at the site of the nucleus of Darkshevitch.

Van Gehuchten concludes that the nucleus of Darkshevitch is an important center for the production of vertical movements of the eyes.

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42. Kelly, D. B.: Deafness and Loss of Vestibular Function Following Herpes of the Scalp, J. Laryng. & Otol. **55**:286-288 (June) 1940.

43. Denny-Brown, B.; Adams, R. D., and Fitzgerald, P. J.: Pathologic Features of Herpes Zoster: Note on "Geniculate Herpes," Arch. Neurol. & Psychiat. **51**:216-231 (March) 1944.

44. van Gehuchten, P.: Syndrome de Parinaud: Etude anatomique, J. belge de neurol. et de psychiat. **40**:126-133 (March) 1940; abstracted, Arch. Neurol. & Psychiat. **45**:345-346 (Feb.) 1941.

Comment (W. P. E).—The sudden tinnitus which ushered in the tegmental hemorrhage probably was caused by the upsetting of the equilibrium of the intracerebral juices which preceded the vascular occlusion in the midbrain, as the nucleus of Darkshevitch is situated in the median portion of the tegmentum of the upper midbrain in the central gray matter adjacent to the tectogeminal fibers. Some of its axons descend in the median longitudinal fasciculus, while other fibers from the nucleus of Darkshevitch cross to the opposite side by way of the posterior commissure. Their location near the reflex nuclei of vision suggests that they are nearly connected with the production of the body's reactions to vision. Cells of this region descend a great distance and are the earliest of all association neurons to send out axons in the embryonic neural tube.[45]

Lesions of the pontile tegmentum cause deafness, and the extensive connections of the nuclei of Darkshevitch could cause a disturbance of the auditory tract, as this tract extends through the midpontile and the upper pontile levels.[46] As far as I am aware, no systematic study has been made to show whether vascular lesions of the tegmentum have been ushered in by tinnitus. Such a research might be profitable. It is certain, however, that slight disturbances of equilibrium in the vascular bed will cause tinnitus.

Deafness in a Case of Encephalomyelitis Optica.—Encephalomyelitis optica (Kennedy [47]) is thought to be an acute infectious disease of the nervous system characterized by involvement of the optic nerve and the upper part of the spinal cord, probably due to a virus (Haddon [48]).

In the case reported by Kennedy, at the end of October 1931 a woman aged 34 experienced malaise and frontal headache, followed in three days by blurred vision in the right eye. Three days later vision in the left eye failed, and within a week she was blind in the right eye and ten days after the onset perception of light was absent in the left eye. Twenty days after the onset she became totally deaf in the right ear and two days later in the left. All symptoms disappeared in a week.

November 18: Shooting pains appeared in the lower extremities, with numbness, tingling and hyperesthesia, and terrific pain in the bladder.

November 28: There was total flaccid paralysis of both legs, with overflow retention of urine, paralysis of the rectal sphincter and loss of pain and temperature sensation. Both arms became flaccid.

45. Krieg, W. J. S.: Functional Neuroanatomy, Philadelphia, The Blakiston Company, 1942, p. 270.

46. See the Rasmussen [8] diagram in Stevens, S. S., and Davis, H.: Hearing: Its Psychology and Physiology, New York, John Wiley & Sons, Inc., 1938, fig. 16, p. 416.

47. Kennedy, F.: Encephalomyelitis Optica, Arch. Neurol. & Psychiat. **48**:855-857 (Nov.) 1942.

48. Haddon, in discussion on Kennedy,[47] p. 856.

December 2: Conjugate movements of the eyes were limited. The neck was stiff. The spinal fluid contained 63 lymphocytes per cubic millimeter and 84 mg. of protein and 40 mg. of sugar per hundred cubic centimeters.

December 8: Complete deafness was recorded for the right ear, and two days later, for the left. At this time there was return of temperature sensation over the trunk, and the Babinski response was observed bilaterally. All reflexes had previously been abolished.

December 13: There was pallor of both nerve heads, and the ocular movements were 75 per cent of normal.

December 16: The patient became lethargic and aphonic, but within three days she was able to talk, heard well with the right ear, moved both legs and the right arm, and perceived light with the right eye. Within a week she could move both legs, sensation returned to the lower extremities and she could hear with both ears.

Three weeks later slurred speech was again noted, and within six hours her speech became unintelligible.

January 20: Speech was clearer. When the patient raised her head from the pillow, she involuntarily jerked it backward and slowly rotated it to the left. This disturbance disappeared after several days.

January 22: Weakness of the right side of the face of the peripheral type was noted.

Two days later she became comatose and both sides of the face were paralyzed. She was again unable to move any of the extremities.

February 3: Spastic cerebral paralysis of the right arm and leg had been imposed on the abating spinal paralysis.

February 7: The optic disks were paper white, and involuntary laughing and crying occurred. Conjugate movements of the eyes returned, but weakness of the right sixth nerve appeared.

Hearing Loss Subsequent to Skull Fracture.—In cases of unilateral longitudinal fracture of the skull the function of hearing is more damaged as age increases (Grove [49]). The ear on the side of the fracture sometimes shows a greater loss of hearing than its fellow, but in a large number of instances the loss is the same in both ears. If deterioration of hearing does not occur in the first six months, the condition will probably remain stationary.

AUDITORY AND FACIAL REFLEX

The Glabella [50] *Reflex* (*Guillain* [51] 1920; *Wartenburg* [52] 1939).—The loss of this nasopalpebral reflex was observed by Asherson as an early sign of impending facial paralysis. (See p. 167.)

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49. Grove, W. E.: The Effect of Head Injury on Hearing, J. Speech Disorders **8**:363 (Dec.) 1943.

50. The glabella is a point that is midway between the two supraorbital ridges.

51. Guillain, G.: Le réflexe naso-palpébral (réflexe trijumeau-facial) et sa valeur pronostique dans la paralysie faciale, Compt. rend. Soc. de biol. **83**:1394-1396, 1920.

52. Wartenberg, R.: A Sign of Facial Palsy, Arch. Neurol. & Psychiat. **41**:586-588 (Feb.) 1939.

Cochleopalpebral Reflex.—Wartenberg [53] points out that the orbicularis oculi muscle protects by contraction the bulbus oculi and the retina from external noxious influences. In the mechanism of its reflex action it shows peculiarities which other muscles do not possess. The reaction of the muscle is rapid—so rapid that in every language there is the expression "quick as a wink." The reaction is bilateral. An outstanding characteristic of the reflex mechanism is that it is brought into play by a great variety of stimuli. The orbicularis oculi muscle contracts on stimulation of the cochlea, the phenomenon being known as the cochleopalpebral reflex or the auropalpebral or acousticopalpebral reflex. This reaction is strikingly manifested when the patient is startled by an unexpected sound, such as a pistol shot (Strauss, Landis and Hunt [54]).

Imperatori's Palatopalpebral Reflex.—Imperatori [55] recently described a palatopalpebral reflex in which the eye closes when the palate is touched.

VESTIBULAR NYSTAGMUS

Cochlear and Vestibular Symptoms Caused by Tumor Situated in the Anterior or the Middle Fossa of the Brain.—Fischer [56] in 1925 and again in 1940 observed that in 77 per cent of cases of tumor situated in the anterior fossa or the middle fossa of the brain there were symptoms of "congestion of the inner ear." The "symptoms of congestion of the inner ear" which he lists may be grouped as follows:

1. Auditory symptoms: (*a*) subjective noises in the head, (*b*) tinnitus, (*c*) diminished hearing, (*d*) shortened bone conduction, (*e*) lowered upper tone limit.

2. Vestibular symptoms: (*a*) dizziness, (*b*) spontaneous nystagmus, (*c*) abnormal irritability of the labyrinths, (*d*) disproportion between the objective labyrinthine reaction and the subjective sensation of dizziness.

3. Marked differences in the aural symptoms present at different examinations. [This is of special significance in my opinion. W. E. P.]

Anatomic Changes in the Temporal Bones from Chronic Increased Intracranial Pressure.—Among the persistent anatomic changes produced by increased pressure of the brain Fischer found (1) hernia of the brain in the region of the normally placed pacchionian granula-

53. Wartenberg, R.: Studies in Reflexes: History, Physiology, Synthesis and Nomenclature; Study I, Arch. Neurol. & Psychiat. **51**:113-133 (Feb.) 1944.

54. Strauss, H.; Landis, C., and Hunt, W. A.: Acoustic Motor Reactions, Arch. Otolaryng. **28**:941-945 (Dec.) 1938.

55. Imperatori, C. J.: The Palatal Palpebral Reflex, M. J. & Rec. **131**:198 (Feb. 19) 1930.

56. Fischer, J.: Changes in the Internal Ear Due to Increased Endocranial Pressure: The Histologic Basis of Congestive Inner Ear, Arch. Otolaryng. **31**: 391-419 (March) 1940.

tions and (2) pressure atrophy of the petrous bones caused by herniation of the brain and of the pacchionian granulations.

Fischer made serial sections of 10 petrous bones from 5 patients who had died of tumor situated in the anterior or the middle fossa of the brain.

(*a*) Hernia of the Pacchionian Grandulations: Three of Fischer's specimens showed hernia in the region where pacchionian granulations are normally located: In 1 the hernia extended into the internal auditory canal (fig. 4 of case 3); in 1 the hernia entered the dura of the middle fossa in the region of the tegmen tympani (fig. 3 of case 2), and in 1 the hernia extended into the aqueductus cochleae (fig. 8 of case 4).

(*b*) Hernia of Brain Extending into the Bone of the Petrous Pyramid: The brain herniated into the bony pyramid of the middle fossa (1) above the semicircular canals, (2) above the canal for the facial and the superficial petrosal nerve and (3) above the roof of the tympanic cavity and the eustachian tube.

Correlation of Pathologic Changes with Clinical Symptoms.—In Fischer's case 1 of endothelioma of the right motor region of the brain the patient had, as aural symptoms, tinnitus (chirping and twitching) and a sensation of fulness in the ears. This was present with normal hearing and normal c and c^{-4} aerial conduction, although the bone conduction was reduced ten seconds.

In this case microscopic section through each cochlea showed the scala vestibuli and the ductus cochlearis "filled with transudate" and hemorrhages in the scala tympani.

Fischer explained the absence of any outstanding hearing defect by the fact that the "congestive process" in the labyrinth had not as yet damaged the delicate sensory epithelium or the nerve ganglion apparatus, for the cells of the "organ of Corti remained intact" although "embedded in transudate."

In spite of there being "choked disks," there existed hyperirritability to unilateral calorization, which I (W. P. E.) think is explainable by the small size and the superficial location of the tumor in the right motor region. This tumor, although it caused "left hemiplegia, paralysis of the left facial nerve, deviation of the tongue to the left, a positive Babinski sign and choked disks," did not produce a great increase in the general intracranial pressure failure of which to react to the cold caloric test in the upright position is a symptom, first described by me in 1912.[57]

57. Eagleton, W. P.: Decompression for the Relief of Disturbances of the Auditory Apparatus of Intracranial Origin, Tr. Am. Otol. Soc. **12**:564-574 1912; Laryngoscope **23**:592-601 (March) 1913.

In case 2, autopsy showed "glioma of the left parietal lobe extending anteriorly into the gyrus centralis anterior" [the ascending frontal convolution, which is the posterior convolution of the frontal lobe]. The auditory symptoms are described somewhat as follows: "The distance at which the accentuated whispered voice could be heard appeared somewhat diminished on both sides." "Diminution of the hearing for the upper tone limit and slight diminution for the accentuated whispered voice occurred." "Bone conduction was slightly shortened." As to aerial conduction, the C fork was well heard, while perception of the C-4 fork was shortened (on the right ten seconds and on the left twelve seconds less than normal).

The following vestibular signs were recorded: There was spontaneous nystagmus to both sides. Bilateral calorization gave negative results, while a unilateral test showed "increased irritability of both labyrinths."

Pathologic Changes in the Inner Ear: In the scala vestibuli and the basal turn of the cochlea there was exudation, with degeneration of sensory cells; also, edema of the interstitial tissue of the spiral ganglion.

Furthermore, in the second turn of the cochlea a small amount of exudate was present, as well as beginning processes of organization causing adhesions between Corti's membrane and the inner pillar of the organ of Corti, with loss of cilia. [This amply explained the hearing loss but not the negative results of bilateral calorization while a "unilateral test showed increased irritability of both labyrinths," which must have been of central origin.—W. P. E.]

Pathologic Facts of Clinical Value Regarding the Effect of Increased Intracranial Pressure on the Inner Ears (Fischer [56]).—1. In brain pressure a destructive pressure may be transmitted into the inner ear by way of the subarachnoid spaces of the aqueductus cochleae, the nerve sheaths in the internal auditory meatus, the pacchionian granulations, the ligamentum spirale or the lymph channels of the bony capsule of the labyrinth [58] and finally by causing "congestion of the blood vessels."

2. Increased generalized brain pressure may exist over a certain period without damaging the delicate sensory epithelium and the nerve ganglion apparatus, even when extensive transudation or exudation is present.

3. Increased generalized brain pressure produces secondary degenerative processes extending from the basal turn of the cochlea to the modiolus when the pressure exists for a long time.

4. Brunner [59] attributed herniation of the brain and edema of the ductus endolymphaticus to generalized brain pressure.

"Nystagmus-Bereitschaft" (Kobrak, 1918-1921) *in the Diagnosis of Cortical Lesions of the Brain.*[60]—The German word *Bereitschaft* means "readiness," "preparedness," "preparation." *Nystagmus-Bereit-*

58. I do not believe there are true lymphatic channels in the bony capsule of the labyrinth (W. P. E.).

59. Brunner, H.: Otologische Diagnostik der Hirntumoren, Berlin, Urban & Schwarzenberg, 1936.

60. Kobrak, F.: Studies on Vestibular Nystagmus, J. Laryng. & Otol. **58**:167-187 (May) 1943.

schaft is a latent form of nystagmus. It is a state of "centrally increased excitability of the vestibular system." It is also called "Nystagmophilia."

[I think "conditioned nystagmus" would be a better term. For it is the "conditioned state" (Pavlov) of the vestibular mechanism that produces the irregularities of the vestibular responses which result from stimulation. The term "nystagmus susceptibility" has been suggested by C. S. Saggit. This "undirectional, dormant nystagmus" is called "directional preponderance nystagmus" by Hallpike, but the correctness of this is disputed on the ground that the term pertains to another form of nystagmus.—W. P. E.]

Kobrak states that "*Nystagmus-Bereitschaft* is not identical with directional preponderance." It is a "tendency to nystagmus which under certain conditions emerges from latency." Its existence "can be deduced from calculated interrelations of figures upon different ways of labyrinthine stimulation."

Kobrak's explanation is: The nystagmus is not elicited through the usual paths via the labyrinth and the nucleus of Deiters. There seem rather to be two alternatives—either (1) activation of latent nystagmus—*Nystagmus-Bereitschaft*—on a retrograde nervous impulse via the vagus-nucleus of Deiters on a cutaneous stimulus, or (2) the interference of vascular reflex conditions, which were suggested by the author as playing a part in the origination of caloric nystagmus in general.[61]

In all of 10 patients who had lesions of one temporal lobe, "directionary predominance of caloric nystagmus was observed to the side of the lesion."[62]

Historical datum: Rosenfeld in 1914 noted that "the direction of nystagmus in cold caloric responses with cortical tumors" is independent of the side of the caloric stimulation.

Fitzgerald and Hallpike obtained results confirming the findings of de Kleijn and Versteegh that cerebral lesions in man may lead to exaggeration of caloric nystagmus to the side of the lesion.

They think that an association of the vestibular system with the temporal lobe, at any rate on the sensory side, is already supported by the anatomic findings of Held (1923). These association fibers convey a clear suggestion of the existence of a tonic controlling action

61. Schmaltz, G.: Versuche zu einer Theorie des Erregungsvorganges im Ohrlabyrinth, Arch. f. d. ges. Physiol. **208**:424, 1925. Dohlman, G.: Physikalische und physiologische Studien zur Theorie des kalorischen Nystagmus, Acta oto-laryng., 1925, supp. 5, pp. 1-196.

62. Cawthorne, T. E.; Fitzgerald, G., and Hallpike, C. S.: Studies in Vestibular Function: Observations on the Directional Preponderance of Caloric Nystagmus ("Nystagmusbereitschaft") Resulting from Unilateral Labyrinthectomy, Brain **65**: 138-160 (June) 1942.

of centers in the temporal lobes, exercised through corticofugal fibers, on the reflex responses of the lower vestibular centers.

Technic of the Caloric Tests to Demonstrate Nystagmus-Bereitschaft.—The stimuli used should be small in order to restrict excitation to the external auditory canal. It is desirable to use hot and cold stimuli which are thermally equidistant from body temperature. Finally, it has been imperative that the conditions of the test should provide maximum delicacy with a view to revealing small differences in excitability of the reflex mechanisms usually involved.

Vestibular Reactions in Schizophrenia.—(*a*) The Genetics of Schizophrenia (Kallmann and Rypins [63]) and Vestibular Reactivity (Angyal Syndrome): Kallmann and Rypins [63] state:

For eugenic purposes it is sufficient that schizophrenia has proven an *organic* disease. [This opinion is based on] a *specific* predisposition which follows the recessive course of heredity and is therefore transmitted to all the children of schizophrenics. It is too often forgotten that only predispositions are inherited and never attributes as such, . . . The children of schizophrenics have . . . a probability of schizophrenia about nineteen times as high as the average population, . . .

Schizophrenia, as well as tuberculosis, is due to "hereditary functional weakness of the reticuloendothelial system."

(*b*) Vestibular Reactivity in the Angyal Syndrome in Schizophrenia: The Angyal syndrome is a certain form of schizophrenia called "loss of ego reference." It has a particular type of dissociation and a constant occurrence of paresthesia, associated with abnormal vestibular reactions.

These schizophrenic patients complain of bizarre changes in the "experience of the body": They have subjective impressions of (1) excessive lightness or heaviness, (2) changes in the size of the body or (3) other distortions of the body. These paresthesias, according to Angyal, are due to dissociation in the muscle tonus. This dissociation in the muscle tonus is a part of the dissociations causing the complex behavior tendencies of these patients.

In 1921 Pekelsky [64] reported 2 cases of catatonic schizophrenia in which there was a *transitory absence of nystagmus in response to vestibular stimulation.*

The purpose of the study reported by Angyal and Blackman [65] was to determine whether the impressions of lightness and heaviness and changes in the size of the body and other distortions of the body image are due to muscular tonus reflected at the level of the involuntary regulation of muscular tonus.

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63. Kallmann, F. J., and Rypins, S. J.: The Genetics of Schizophrenia, New York, J. J. Augustin Publishers, 1938; reviewed, Arch. Neurol. & Psychiat. **43**: 857-858 (April) 1940.

64. Pekelsky, A.: Transitorischer Anystagmus bei Katatonie. Ist der Nystagmus willkürlich unterdruckbar? Rev. v neuropsychopath. **18**:97-102, 1921; abstracted, Zentralbl. f. d. ges. Neurol. u. Psychiat. **26**:291, 1921.

65. Angyal, A., and Blackman, N.: Vestibular Reactivity in Angyal Syndrome in Schizophrenia, Arch. Neurol. & Psychiat. **44**:611-620 (Sept.) 1940.

Angyal and Blackman based their study on reactions to turning and caloric stimulation in 58 male schizophrenic patients as contrasted with the reactions observed in 20 normal persons.

Angyal and Blackman concluded: 1. The response of the patients to rotatory stimulation was significantly lower than that of the normal controls; the mean of the number of nystagmic beats in the schizophrenic group was 44.9 (normal 60.8) and the mean of the frequencies was 1.32 (normal 1.68).

2. In response to caloric stimulation the difference between the schizophrenic patients and the normal controls was even greater; the absolute number of nystagmic beats in the schizophrenic group being 60.2 (normal 116.4) and the mean of the frequencies 0.82 (normal 1.34).

3. The patients with the syndrome described by Angyal (5 in all) exhibited the lowest reactivity to both caloric and rotatory stimulation.

4. In the case of a patient with a periodic course alternating between stupor and relative freedom from symptoms there was considerable parallelism between the vestibular reactivity and the clinical state.

5. The degree of reduction of the vestibular reactivity was not correlated with the conventional subtypes of schizophrenia.

(*c*) Spontaneous Nystagmus and Absence of Induced Nystagmus in Catatonic Schizophrenia: Rosenfeld[66] in 1926 observed spontaneous nystagmus in 4 patients with catatonic schizophrenia. Claude, Baruk and Aubry[67] studied a group of 12 patients with schizophrenia (4 with catatonia); 2 failed to show nystagmus in response to caloric and galvanic stimulation; the third showed a markedly diminished, and the fourth, a slightly diminished reaction.

Nystagmus in Hysteria.—Nystagmus not infrequently occurs in cases of hysteria, and it may be produced voluntarily by the hysterical person. Hysterical nystagmus, unlike the oscillating nystagmus caused by an organic lesion, is jerky and irregular.[68]

Cerebellar Type of Vertical Nystagmus in Cerebellar Lesions Not Due to Involvement of the Vestibular Nuclei.—Spiegel once believed that nystagmus in cerebellar diseases was due either to pressure of an abscess on the vestibular nuclei or to infiltration of these nuclei by the infecting organisms, but his view is now changed. For nystagmus

66. Rosenfeld, M.: Ueber das Vorkommen von Nystagmus bei Schizophrenia, Deutsche med. Wchnschr. **52**:985-988 (June 11) 1926.

67. Claude, H.; Baruk, H., and Aubry, M.: Contribution à l'étude de la démence précoce catatonique: Inexcitabilité labyrinthique au cours de la catatonie, Rev. neurol. **1**:976-980 (June) 1927.

68. Bing, R.: Textbook of Nervous Diseases, St. Louis, C. V. Mosby Company, 1939, chap. 30, p. 779; chap. 2, p. 93.

may appear with cerebellar lesions which leave the vestibular nuclei and fiber systems histologically intact.

(*a*) Positional Nystagmus: Spiegel has demonstrated that in the normal position no nystagmus appears but that with a change of position of the whole body and head, nystagmoid movements are elicited.

Experiments on cats by Spiegel and Scala [69] showed that a cerebellar type of vertical nystagmus occurs. This vertical nystagmus is other than that produced by lesions of the vestibular nuclei. This cerebellar form of vertical nystagmus was observed after lesions of the vermis, with the vestibular nuclei and their fiber system histologically intact.

The positional nystagmus observed after a paleocerebellar (vermis and flocculus) lesion probably belongs in the group of symptoms of overexcitability of the vestibulo-ocular reflex arc that follow such a cerebellar lesion.

(*b*) Cephalogyric Reactions (Circular Movements of the Head) After Section of the Eighth Nerve: According to Spiegel,[70] bilateral section of the eighth nerves after galvanic stimulation of the labyrinth in decerebrate cats reverses the direction of the rotation of the head produced by a direct current. After unilateral section the rotation to the anode is usually preserved when the cathode lies on the normal side, while the rotation to the cathode may appear if the latter lies on the side operated on. Spiegel states:

The centers of the ipsilateral cephalogyric reaction lie in oblongata and pons. The reaction elicited by stimulation of afferent trigeminal fibers uses efferent fibers descending into the spinal cord; an efferent pathway along the accessory [nerve] could not be established with certainty for this part of the reaction. For the ipsilateral rotation elicited by stimulation of afferent 9th-10th fibers, efferent pathways could be demonstrated using the accessory nerve as well as fibers descending into the spinal cord.

(*c*) Experimental Studies of Positional Nystagmus in Animals: Little is known regarding the influence of the cerebellum on vestibular reflex arcs as they extrude through the oblongata and the pons (Spiegel and Scala [71]).

The experiments of Spiegel and Scala demonstrated that after lesions of parts of the cerebellum of cats, particularly of the lobus posterior medianus, "nystagmus was observed in abnormal positions of the head, particularly in the supine position with the vertex downward. . . . Bilateral labyrinthectomy abolished this effect of position, . . . It is assumed that the appearance of positional nystagmus following

69. Spiegel, E. A., and Scala, N. P.: Vertical Nystagmus Associated with Cerebellar Lesions, Arch. Neurol. & Psychiat. **47**:192-193 (Jan.) 1942.

70. Spiegel, E. A.: Cephalogyric Reactions of Nonlabyrinthine Origin, Am. J. Physiol. **135**:628-632 (Feb.) 1942.

71. Spiegel, E. A., and Scala, N. P.: Positional Nystagmus in Cerebellar Lesions, J. Neurophysiol. **5**:247-260 (July) 1942.

cerebellar lesions is a phenomenon of release of parts of the vestibulo-ocular reflex arcs, since it is associated with increase of the experimental postrotatory nystagmus." The experiments suggest that routine tests should be made for positional nystagmus in patients suspected of having cerebellar lesions.

A lesion of the vermis cerebelli–nuclei tecti system may produce in some species transient overexcitability of these reflex arcs. Thus, in rabbits there was an increase in the duration and the intensity of experimental nystagmus (Bauer and Leidler [72]), and in decerebrate cats there was an increase of tonic labyrinthine reflexes on limb and trunk muscles (Pollock and Davis [73]). However, after subtotal ablation of the uvula and the modiolus in monkeys and chimpanzees, negative results were reported by Dow.[74]

Electric Stimulation of the Afferent Neurons of the Vestibular Apparatus.—Electric stimulation of the labyrinth [75] is rather neglected [76] because it acts not only on the receptor organ but also on the afferent neuron (vestibular ganglion—Dohlman [77]).

The spontaneous nystagmus occurring after unilateral labyrinthectomy and also Bechterew's compensatory nystagmus show periodic increase and decrease (Spiegel and Yaskin).

Spiegel [76] in attempting to interpret his experiments raises the question whether the reactions to rhythmic stimulation are due to excitation of the vestibulo-ocular reflex arc or result from direct stimulation of the ocular muscles or of their motor neurons.

Technic.—The connections between one labyrinth and the nuclei of the ocular muscles were severed by an incision on the inner side of the restiform body, and the concentric needle electrode was inserted into the homolateral labyrinth. Rhythmic electric stimulation of this labyrinth failed to produce ocular reactions.

The fact that distinct reactions to rhythmic stimulation of the labyrinth can be produced only at relatively low frequencies probably is related to the relatively high chronaxia of the vestibular nerve.

Rates of stimulation which were apparently inadequate to produce definite ocular reactions by acting on the vestibulo-ocular reflex arc,

72. Bauer, J., and Leidler, R.: Einfluss der Ausschaltung verschiedener Hirnabschnitte auf die vestibulären Augenreflexe, Arb. a. d. neurol. Inst. a. d. Wien. Univ. **19**:155-226, 1912.

73. Pollock, L. J., and Davis, L.: Influence of the Cerebellum upon the Reflex Activities of the Decerebrate Animal, Brain **50**:277-312 (Oct.) 1927.

74. Dow, R. S.: Effect of Lesions in Vestibular Part of Cerebellum in Primates, Arch. Neurol. & Psychiat. **40**:500-520 (Sept.) 1938.

75. The term "labyrinth" as used here covers not only the receptor cells of the peripheral sense organ but also the afferent neuron.

76. Spiegel, E. A.: Rhythmic Stimulation of the Labyrinth, Arch. Otolaryng. **33**:572-578 (April) 1941.

77. Dohlman, G.: Experimentelle Untersuchungen über die galvanische Vestibulärisreaktion, Acta oto-laryng., 1929, supp. 8, p. 1.

however, sometimes influenced nystagmus caused by injury to the inner ear. In analyzing the effects of the experiments one must bear in mind that rather strong currents had to be applied in order to produce the reactions recorded. Strong stimulation of sensory nerves, such as the sciatic, can also increase the amplitude and frequency of an existing weak spontaneous nystagmus. Radiation of impulses from other sensory nerves and pathways to the vestibulo-ocular reflex arc seems therefore to play an important part in the genesis of the effects on spontaneous nystagmus described here.

Bárány, in 1907, presented a brief outline of an improved method of examining for galvanic nystagmus of each ear separately and cited Neumann's experiment in which the eighth nerve was galvanized after labyrinthectomy.[78]

In deaf-mutes in Vienna, Dr. G. W. MacKenzie found the reactions positive, whereas in patients with meningitis no reactions were elicited. The galvanic test served him faithfully in differentiating bilateral neurolabyrinthitis syphilitica, with absence of galvanic reactions, and bilateral labyrinthitis syphilitica tarda, in which the galvanic reactions are normal.

The fact that section of the eighth nerve medial to Scarpa's vestibular ganglion causes degeneration of that nerve with consequent absence of responses to electrical stimulation, does not coincide with the fact that after labyrinthitis, which is a lesion peripheral to Scarpa's ganglion, there exists some response at first but the reactions disappear. This seems to indicate that degeneration takes place even when the lesion is peripheral to the ganglion.

Dr. Julius Winston stated:

> . . . Electrical stimulation, save for its ability to differentiate between a lesion in the semicircular canals and one of the nerve proper, has little clinical application. . . . one finds that frequently with caloric stimulation the vertical canals are not responsive while the horizontal canal reacts normally. It has been pointed out that the electric stimulus stimulates the end organs in all the semicircular canals, and so does not reveal absence of function of the vertical canals.

Dr. Spiegel stated:

> . . . Clinical usefulness of the galvanic test is limited chiefly to the differential diagnosis of labyrinthine and retrolabyrinthine lesions. If several weeks elapse after extirpation of the peripheral labyrinth, destruction of the peripheral endings of the vestibular nerve induces secondary degeneration of the cells of the ganglion vestibulare Scarpae and, consequently, the vestibular nerve loses its excitability to galvanic stimuli, so that the primary lesion of the labyrinth is followed by the picture of retrolabyrinthine disease due to the secondary degeneration of the neuron of the vestibular nerve. . . . [The symptom of] the so-called lack of

78. Spiegel, E. A., and Scala, N. P.: Response of the Labyrinthine Apparatus to Electrical Stimulation, Arch. Otolaryng. **37**:746-747 (May) 1943; **38**:131-138 (Aug.) 1943.

excitability of the vertical canals in cases of increased intracranial pressure, . . . discovered by Eagleton,[57] is of high diagnostic importance. In interpreting it, one has . . . to bear in mind that in these cases the vertical canals are still excitable for rotational stimuli, as was first emphasized by the French author Hautant. The caloric stimulus is a rather weak stimulus for the vertical canals; thus they easily fail to respond to calorization if the reactivity of the labyrinth is impaired, while the stronger rotational stimuli may still reveal excitability.

Positional Nystagmus and Vertigo.—Bárány [79] described nystagmus and vertigo occurring only with certain positions of the head: The patient showed these reactions when in the dorsal recumbent position, with the head turned to the right side, so that the left labyrinth was up and the right labyrinth down.

The patient also showed nystagmus and dizziness in the sitting position whenever the head was tilted so that the right labyrinth was on top and the left labyrinth beneath. Bárány designated the nystagmus as "positional nystagmus" because it was dependent on a definite position of the head. He assumed that it was elicited by the otolithic apparatus (otolithic reflex). Brunner and Stein, also Grahe and others, expressed the belief that central lesions, as of the neck reflexes (Magnus and DeKleijn), were responsible.

Cerebral Origin of Airsickness During Acrobatics.—During any movement of the body, the vestibular apparatus sends impulses to the brain. Flaherty [80] states:

When the vestibular apparatus becomes overstimulated and the brain can no longer interpret these impulses properly, the person soon becomes nauseated, and vomits. . . . It becomes imperative that a person interpret these sensations properly or ignore them in part in order to prevent airsickness.

. . . So long as a person can orient himself in space by visual means, he can ignore or modify impulses coming from the vestibular apparatus.

During blind flying when the eyes can no longer tell the pilot his position in space, even the most experienced of pilots have the sensation of vertigo . . . they must depend entirely upon their instruments to tell them that the sensations received from the vestibular apparatus are false.

The same holds true, in most cases, for the student engaged in acrobatics.

Typical Syndrome of Tumor of the Cerebellopontile Angle.—Winston's [81] analysis of 34 cases of verified tumor of the cerebello-

79. Bárány, R.: Die nervoesen Stoerungen des Cochlear und Vestibular Apparatus, in Lewandowsky, M.: Handbuch der Neurologie, Berlin, Julius Springer, 1910. Fischer, J., and Wolfson, L. E.: The Inner Ear, New York, Grune & Stratton, Inc., 1943, p. 59.

80. Flaherty, T. T.: Airsickness During Acrobatics, U. S. Nav. M. Bull. **40**:902-906 (Oct.) 1942; abstracted, Neuro-Psychiatric Institute of the Hartford Retreat, Oct. 30, 1942.

81. Winston, J.: A Revision of the Cerebellopontile Angle Lesion Syndrome with an Analysis of the Vestibular Findings in Thirty-Four Cases of Verified Tumor of the Cerebellopontile Angle, Arch. Otolaryng. **32**:877-886 (Nov.) 1940.

pontile angle suggests that the typical syndrome as it is generally understood appears frequently with both neural and extraneural tumors of the cerebellopontile angle (the former arising primarily from the eighth nerve; the latter involving the eighth nerve secondarily).

Practically all of Winston's patients had (1) a spontaneous nystagmus when looking up, down or straight ahead, (2) a disproportion between the duration of the induced nystagmus and that of the vertigo after turning and (3) a loss of function of either the vertical semicircular canals or the vertical and the horizontal semicircular canals. The vestibular symptoms were associated with (4) good hearing on the side opposite the lesion.

Tolerance to Vestibular Stimulation: All of Winston's patients showed tolerance to vestibular stimulation. In 7 whose tumor was unilateral the opposite vertical and horizontal semicircular canals functioned normally.

Winston states:

> The cerebellopontile angle lesion syndrome was typically demonstrated in 59.2 per cent of the cases of unilateral angle tumor. The term does not apply . . . when a bilateral angle lesion is present. If the designation of the syndrome were changed to "evidence of a peripheral lesion on the same side as the cerebellopontile angle lesion in addition to evidence of a central lesion," then 85.3 per cent of the 34 cases of this series could be said to conform . . . to the revised concept of the syndrome associated with a lesion of the cerebellopontile angle.

Winston accepts Lewis' definition:

> A single lesion at or near the junction of the eighth nerve and brain stem frequently causes total abrogation of function of the cochlea, the horizontal and vertical canals of the same side, and the vertical canals of the opposite side. This is called the cerebellopontile angle lesion syndrome.

He also accepts the statement of Grant and Fisher:

> In a cerebellopontile angle lesion, the only portions of the ear mechanism functioning properly are the cochlea and the horizontal canal of the good or unaffected side.

Comment (W. P. E.) (Influence of Increased Intracranial Pressure on the Result of the Cold Caloric Test).—The confusion regarding the interpretation of the vestibular signs in the patient with tumor of the cerebellopontile angle is illustrated by Winston's report. Neither Lewis' nor Fisher's concept recognizes the influence of the increased intracranial pressure as a cause of absence of reaction on the side opposite to that given the cold caloric test with the patient in the upright position.

If the abnormal vestibular findings are analyzed on the basis of (1) the presence or (2) the absence of increased intracranial

pressure,[82] and if (3) the abnormalities in the reaction are due not to the involvement of special semicircular canals but to abnormal functional alteration of the whole vestibular nervous system, the vestibular tests are much more understandable and are of great diagnostic value.

VERTIGO

The best psychologic interpretation of vertigo that has appeared in the literature in recent years was contributed by Brain in 1938. When this work is studied in conjunction with Panse's [83] dissertation on the anatomic pathways that are involved in vertigo many of the clinical problems presented by this symptom become explainable.

Central Vertigo.—(*a*) Mathematical and Psychophysiologic Aspects of Vertigo (Brain [84]); Hallucination of Movement: "There is a field of conscious phenomena which are perceived as localized in space." Within this field one can psychologically distinguish two schemata: One represents that portion of space which is occupied by the body, as distinguished from the rest of space. This schema is constantly changing—a "plastic schema," to use Head's [85] phrase. The external world is represented by another schema, also constantly modified. These two schemata, however, are far from independent of each other: The bodily plastic schema receives contributions from proprioceptors (one of the peripheral end organs of the afferent nerves, present in the labyrinth, the muscles, the tendons and other structures, which are in part stimulated by an external force, gravity). On the other hand, the perception of external space includes in its material proprioceptor impulses. The two schemata therefore to some extent interpenetrate and must be conceived as "functional," not "structural," units. For consciousness, the orientation of the body in space is normally an orderly dynamic relation between the bodily schema and the schema of the external world. "Vertigo is the state of consciousness which arises when this relation becomes disordered."

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82. Eagleton, W. P.: The Importance (1) of the Absence of Reactibility in the Vertical Canals of Both Ears to Stimulation by the Cold Caloric in the Diagnosis of Increased Intracranial Pressure of the Posterior Fossa, and (2) of the Reduction of the Duration of the Nystagmus from Turning in Diffuse Lesions of the Cerebrospinal System Pathways over the Cerebellar Cortex (as in Protective Meningitis and Linear Fractures of the Skull of the Posterior Fossa), Tr. Am. Laryng., Rhin. & Otol. Soc. **28**:231-284, 1922.

83. Panse, R.: Vertigo, Arch. f. Ohrenh. **75**:69-75, 1908; translated by A. Knapp, Arch. Ophth. **31**:467-520, 1902.

84. Brain, W. R.: Vertigo: Its Neurological, Otological, Circulatory, and Surgical Aspects, Brit. M. J. **2**:605-608 (Sept. 17) 1938.

85. Head, H., and others: Studies in Neurology, London, H. Frowde, 1920, vol. 2, p. 605.

Two illustrations help to make this clear. First, the simplest form of vertigo is that which is produced by paralysis of an ocular muscle [86]; in this instance, because of faulty projection of the visual field of the affected eye, the patient is presented with two mutually conflicting visual spaces.

The second illustration deals with the important part which *hallucination of movement* plays in the symptom complex of vertigo.[87] The perception of relative movement between the body and the external world must depend on a certain functional relation between the bodily schema and the schema of the external world.

This occurs when a rotation of the visual field causes a hallucination of the body as moving in the opposite direction although the subject of the hallucination is stationary. A familiar example of this type of hallucination is the experience of a person seated in a train. When a train beside his own begins to move, he feels that his own train is moving in the opposite direction.

Thus whether a perception of relative movement between the body and the external world is referred to the body or to the external world depends not only on the content of visual perception but also on the state of the bodily proprioceptors at the time, especially the proprioceptors of (1) the ocular muscles and (2) the labyrinths.

If, owing to acute labyrinthine disease, such as hemorrhage or fracture, a patient feels as if his body is in violent motion in a certain direction, and if nevertheless the objects at which he is looking remain in front of his eyes, he will experience the hallucination that the external world is moving in the direction in which he feels himself to move; for, although he feels himself to be moving, there is no cortical process corresponding to relative movement between his body and the external world. Consequently, the external world is "perceived" as moving in the same direction as his body feels to him to be moving.

(*b*) Principle of Relativity in Vertigo; the Abstract Personal Space from Which Hallucinations of Movement Are Perceived: The fact that a person can experience a hallucination of rotation of both his body and the external world in the same direction implies a space in reference to which they—the body and the external world—are perceived as rotating, which space itself remains stationary. It appears, therefore, that both (1) the schema of the body and (2) the schema of the external world are built up within (3) an absolute space, which absolute or, better named, abstract space is conditioned by one's sensory organs and nervous system. This personal abstract space forms a frame of reference within which the whole visible world, including the body, may be "perceived" as moving. This purely personal absolute space is one of the obstacles which the nonmathematical student encoun-

86. Taylor, J.: Selected Writings of John Hughlings Jackson, London, Hodder & Stoughton, 1932, vol. 2, p. 303.

87. Hartmann, G. W.: Gestalt Psychology, New York, The Ronald Press Co., 1935, p. 249.

ters in trying to grasp Einstein's conception that all movement is relative.

(*c*) Brain's Classification of Clinical Types of Vertigo: Brain recognized five types of intracranial vertigo: (1) vertigo due to disturbances of the cerebral cortex, (2) vertigo of cerebellar origin, (3) vertigo due to lesions of the brain stem, (4) vertigo due to lesions of the eighth nerve,[88] (5) "psychogenic vertigo," as well as the well known (6) aural (labyrinthine) vertigo and (7) vertigo of ocular origin.

In the transitory diplopia which may occur without loss of consciousness, the two images are seen one above the other. The diplopia is due to skew deviation of the eyes (a disorder of ocular posture emanating from the labyrinth and sometimes occurring transitorily after section of the auditory nerve, as H. Cairns and W. R. Brain have shown). Treatment of Aural Vertigo by Section of the Auditory Nerve, *Lancet* **1**:946-952 [May 6] 1933.

Focal etiologic factors of vertigo include infections of the teeth, the tonsils or the nasal sinuses. Brain agrees with Wright that focal sepsis is the most important single etiologic factor.

Other articles bearing on this subject are:

Eagleton, W. P.: Intradural Conditions in Relation to Rhinology and Otology, Arch. Otolaryng. **26**:576-578 (Nov.) 1937.

Collet, F. J.: Le phénomène de la "joue morte" dans le vertige de Ménière, Rev. d'oto-neuro-opht. **13**:665-671 (Nov.) 1935.

Shapiro, S. L.: Vertigo as a Syndrome in Vascular Diseases, Illinois M. J. **70**:512-516 (Dec.) 1936.

Wright, A. J.: Aural Vertigo, J. Laryng. & Otol. **53**:97-112 (Feb.) 1938; Labyrinthine Giddiness: Its Nature and Treatment, Brit. M. J. **1**:668-670 (March 26) 1938.

(*d*) Cortical Vertigo: Since dizziness is a state of consciousness in which spatial elements play an important part, mediated by the cerebral cortex even when it is the outcome of a disorder at a lower level, it follows conversely that a disorder of the cortex may cause vertigo.

Foerster[89] observed that "stimulation of the superior lip of the interparietal sulcus" (Turner's sulcus[90]) evokes violent vertigo. The patient either sees the objects before him moving toward the side of

88. Vertigo due to abnormal vessels pressing on the eighth nerve has been described by Holmes (1924) and W. E. Dandy (Pathologic Changes in Ménière's Disease, J. A. M. A. **108**:931-938 [March 20] 1937).

89. Foerster, O.: The Motor Cortex in Man in the Light of Hughlings Jackson's Doctrines, Brain **59**:135-159 (June) 1936.

90. The sulcus of Turner—sulcus interparietalis—is a branchiofissure starting from near the sylvian fissure at the posterior border of the posterior central gyrus and running obliquely backward and medially, to terminate in the transverse occipital sulcus; it separates the superior and inferior parietal lobes. (Stedman, T. L., and Garber, S. T.: Stedman's Practical Medical Dictionary, ed. 15, Baltimore, William Wood & Company, 1942.)

stimulation or has the sensation of turning toward the opposite side—this despite the fact that in reality no turning of the head and the trunk occurs.

Thus electrical stimulation of a cortical level may evoke a hallucination of rotation—either of rotation of the body in one direction or of rotation of the environment in the opposite direction. Which scheme is felt to be moving and which is felt to be stationary probably depends on the preexisting background provided by the state of the proprioceptors of the body.

(*e*) Cerebellar Vertigo: Vertigo of cerebellar origin occurs especially when the lesion involves the inferior vermis, which is closely linked anatomically with the vestibular central ganglionic system.

This presumably is the cause of the vertigo occurring at the onset of thrombosis of the posterior inferior cerebellar artery.

(*f*) Vertigo Due to Lesions of the Brain Stem: Brain states that "vertigo may be caused by vascular or neoplastic lesions of the brain stem but is most strikingly seen when a patch of disseminated sclerosis involves the pons."

(*g*) Psychogenic Vertigo: This type, according to Brain, is usually associated with (1) severe feelings of anxiety ("I am going to fall if I go out"; "I am walking on air") and (2) symptoms of overactivity of the sympathetic nervous system. He states:

. . . At the psycho-physiological level neurotic vertigo can perhaps best be explained as the effect of bringing into consciousness elements in spatial orientation (the relation of the body schema to the external world schema) which are normally unconscious, the cause . . . being that this physical relationship has become for the patient symbolical of a psychological relationship between himself and his environment which is a source of difficulty to him. Such a process of symbolization is well illustrated by the double (physical and moral) meaning of the words "lapse," "fall," "fallen," and "downfall." Vertigo may also occur as a conversion symptom in hysteria.

Vestibular Vertigo: (*a*) Clinical Differences Between Central and Peripheral Types: Grove [91] thinks that the explanation of the central type of vertigo lies in vasomotor instability, either generalized or restricted to specific areas. A change in the vasomotor control causes an increase in capillary permeability, and this results in localized areas of stasis and edema, the end result of which is destruction of brain tissue and irritation of the central vestibular nuclear masses. Late petechial hemorrhages may well result from the increased capillary permeability.

(*b*) Differences in the Functions of Vestibular Pathways in Man and Animals as Demonstrated by Sectioning Auditory Nerves: The

91. Grove, W. E.: Vertigo, Wisconsin M. J. **40**:799-805 (Sept.) 1941.

effects produced by sectioning one or both auditory nerves in the dog and the cat are different from those observed in man, according to Dandy and Kunkel.[92]

In the cat and the dog, section of an auditory nerve results in frequent spells of violent whirling with rolling to the side of the sectioned nerve. On the other hand, section of both auditory nerves results in violent whirling with turning to either side. Removal of one half of the cerebellum does not produce rotary movements; removal of the entire cerebellum does not cause whirling.

In man after sectioning of a vestibular nerve the body itself does not rotate as in animals. But with the eyes open the effect is that of a hallucination of *rotation of objects* in the room (Hartmann [87]). With the eyes closed, the sensation is one of rotation of the body. For in man the aberrations of vestibular function after sectioning are transferred to the visual tracts, the effect produced being subjective and visual; on the other hand, in animals the aberration is somatic and objective. Doubtless this transfer of the vestibular function to the visual tracts is related to the lessened importance of the semicircular canals of man as compared with those of animals.

Dandy and Kunkel state that section of both eighth nerves in man has little, if any, effect on the equilibrium of the body, the effect being a jumbling of the appearance of objects when the body is in motion; i. e., the effects are on ocular coordination. These physiologic effects, therefore, indicate the probability that the vestibular pathways in human beings pass up the brain stem to the centers controlling the extraocular movement, while those in dogs and cats pass predominantly to the cerebellum.

15 Lombardy Street.

92. Dandy, W. E., and Kunkel, P. A.: The Central Connections of the Vestibular Pathways, Am. J. M. Sc. **198**:149-155 (Aug.) 1939; abstracted, Arch. Neurol. & Psychiat. **43**:374 (Feb.) 1940.

(To Be Continued)

Abstracts from Current Literature

Ear

HEARING LOSS IN CEREBRAL PALSIED CHILDREN. BERNEICE R. RUTHERFORD, J. Speech Disorders **10**:237, 1945.

One hundred and seventy-five children enrolled at the Dowling School for Handicapped Children, Minneapolis, were given audiometric tests. Three classifications of these children were made: i. e., those with cerebral palsy, those with congenital physical defects and those with acquired physical defects. The records of 7 were discarded as being unsatisfactory. In the remaining 168 children there were hearing losses in 15 per cent of the entire number with good speech and in 17 per cent of the entire number whose speech was defective. The group with cerebral palsy was more closely comparable to the group with congenital physical defects than to the group with acquired physical defects in incidence of hearing loss. Fourteen per cent of the group with cerebral palsy, 11 per cent of the group with acquired defects, and 0.6 per cent of the group with congenital defects had severe hearing loss. The percentage of children showing hearing loss is greater in the congenital defect good speech subgroup, 21 per cent; a comparison of the defective speech subgroups showed the percentage with hearing loss to be greater in the cerebral palsy defective speech group, 29 per cent. The incidence of hearing loss is greater in the extrapyramidal (or athetotic) defective speech subgroup than in the pyramidal (or spastic) defective speech subgroup of the cerebral palsy group. There seems to be evidence in the reactions of these children handicapped by cerebral palsy that all of the hearing problem is not due to the hearing loss per se but is part of the larger picture of cerebral palsy.

PALMER, Wichita, Kan.

ENDAURAL MASTOIDECTOMY—FIVE YEARS' EXPERIENCE. GORDON J. MCCURDY, Laryngoscope **55**:349 (July) 1945.

The endaural approach for mastoidectomy is described. The ease with which it can be done is emphasized. Except for certain parts of certain mastoid bones, exposure is adequate. One hundred and twenty-two cases in which this approach was used make up the series reported. The average length of hospitalization was five days. Local anesthesia was found to be better than general for adults.

McCurdy lists the disadvantages and the advantages of this procedure for simple mastoidectomy. He finds no disadvantages in performing the radical and the modified radical mastoidectomy. He feels that the advantages clearly overweigh the disadvantages in all types of mastoidectomy.

Outlines of his cases are given.

HITSCHLER, Philadelphia.

TREATMENT OF "SURGICAL MASTOIDITIS" WITHOUT OPERATION. M. JOEL, Acta oto-laryng. orient. **1**:70, 1945.

This is a translation from the French original. It is unique in recommending that, instead of operating, one may press on the mastoid region and push the pus over into the middle ear, whence it may drain through the auditory canal—a sort of "reverse English." The first to report this procedure seems to have been some old provincial doctor in the Balkans (perhaps he did not possess a knife) who "cured" a peasant without operation. The finding was obviously a subperiosteal abscess, which the early otologists used to cure by "Wilde's incision." Joel reports 2 cases. One of the patients had undergone mastoidectomy at Tel-

Aviv, Palestine. Although adenoids were diagnosed, no mention is made of their removal. Recurrence took place in both ears. A "cross incision" was made in the tympanic membrane, and the pus was forced out through the canal by thumb pressure! By the fourth day, the patient appeared cured. Joel says that this method makes operation superfluous. One wonders whether biologists might consider this a "reversion to ancestral type" or perhaps a reversion to the days antedating the barber surgeon.

VOORHEES, New York.

A CONTRIBUTION TO THE PROBLEM OF INTRACRANIAL COMPLICATIONS OF LABYRINTHINE ORIGIN. ALFRED LASKIEWICZ, Acta oto-laryng. orient. **1**:81, 1945.

Laskiewicz says that in the majority of cases an intracranial complication is due to an infection spreading from the middle ear through the round window to the internal ear (labyrinth) and thence through the internal auditory canal to the brain or the meninges. Thrombi of labyrinthine blood vessels are present as a rule, and Streptococcus mucosus or Diplococcus lanceolatus is the offending organism. Cholesteatoma favors circumscribed labyrinthitis, which often involves the horizontal canal, with resultant fistula. The author "masters" abscess of the internal auditory canal by establishing a wide communication between the canal and the vestibule, with subsequent drainage of Trautmann's triangle. If facial paralysis supervenes, it may subside. The sulfonamide drugs are a great "support" to surgical treatment of the labyrinth. A final note on the use of penicillin seems to be anticipatory and not based on experience at the time the paper was written.

VOORHEES, New York.

Larynx

LATE CLINICAL AND PHONETIC RESULTS OF THE DEVELOPMENT OF LARYNGEAL PAPILLOMAS IN CHILDREN. WALTHER IMFELD, Pract. oto-rhino-laryng. **5**:54, 1943.

Imfeld reports on postoperative control of laryngeal papillomas in children. These growths are both clinically and phonetically more unfavorable in behavior than any other benign growths. Those occurring in the first three years of life usually have a graver prognosis, because of more frequent recurrences than those that occur about puberty, which usually lose their tendency to recur. Many of these patients have shown a permanent injury of speech due to the anatomic extensions and involvements.

PERSKY, Philadelphia.

DIPHTHERIA OF THE LARYNX AND TRACHEOTOMY. K. BERNFELD, Acta oto-laryng. orient. **1**:73, 1945.

This paper was read before the Oto-rhinological and Pediatric Society in Jerusalem in 1943. Bernfeld reports 4 cases which he picked out from an experience of fifteen years in order to set forth "what may be expected after tracheotomy has been performed for relief of diphtheria of the larynx." The old diagnostic indecision between tonsillitis and diphtheria seems to have been again a puzzle. The author thinks it questionable whether "the diphtheric nature of tonsillitis can be established in the absence of positive bacterial findings." He says that this cannot be confirmed in some cases until one performs tracheotomy and cultures material removed from the lower airways. In any kind of "stridor," therefore, he advises antidiphtheritic treatment "even if occasionally the bacterial findings may later be negative." In the United States this has been the rule, one assumes, for many, many years. To do less is to subject the child to the risk of fatality, which is inexcusable and, one may say, criminal.

VOORHEES, New York.

PROBLEMS CONNECTED WITH THE CLINICAL ASPECT AND THE THERAPY OF CANCER OF THE LARYNX. ALFRED LASKIEWICZ, Acta oto-laryng. orient. **1**:85, 1945.

This is the concluding lecture on the subject. The reviewer does not have access to the foregoing dissertations. Two methods of management are at hand, conservative and radical. The former embraces thyrotomy, thyrocricotomy, hemilaryngectomy and partial anterior laryngectomy (Chevalier Jackson), which is limited to cancers of the anterior part of the larynx. Billroth is credited with devising total laryngectomy, which is, of course, the radical method. The choice depends on the location and the size of the tumor, the presence or the absence of metastases, the patient's age and general physical condition and the histologic diagnosis. Cancer at the commissure of the vocal cords should be treated by radium, especially if the patient will not consent to surgical treatment. One should take into account the roentgen resistance of some tumors. The technic and the dosage are described. Subcordal cancers are not responsive to roentgen therapy. It is better to do hemilaryngectomy or laryngofissure with subtotal removal. The author quotes the "results" (cures?) of St. Clair Thomson as 88 per cent. Complete laryngectomy is contraindicated if cancer is already evident in the upper part of the esophagus and the surrounding areas, if there are large lymph nodes in the neck and the mediastimum with large blood vessels and probably uncontrollable hemorrhage, if the growth involves the base of the tongue and beyond, and if tuberculosis, diabetes or renal disease is present. There is much more which will well repay a careful reading.

VOORHEES, New York.

Miscellaneous

STUDIES ON THE CONTROL OF ACUTE INFECTIONS OF THE RESPIRATORY TRACT. MORRIS SIEGEL, Am. J. Dis. Child. **70**:89 (Aug.) 1945.

This is the fourth communication which stems from the division of infectious diseases of the Public Health Research Institute of the City of New York, aided by a grant from the Metropolitan Life Insurance Company. The study was undertaken to determine the effect of sulfadiazine when used as a prophylactic to reduce the incidence and the severity of infections of the respiratory tract in physically normal children. During the period between August 1942 and April 1944 sulfadiazine was given to 128 such children over periods of four to fifteen consecutive weeks in daily doses of 0.5 to 2 Gm.. The average blood level obtained was 3.5 mg. per hundred cubic centimeters with a maintenance dose of 1 Gm. a day and 7.2 mg. per hundred cubic centimeters with 2 Gm. a day. This therapy was not discontinued even in the presence of serious toxic reactions. The incidence of acute infections of the airways was determined to be about the same in treated and in untreated children, but it seemed that the treated ones recovered more promptly than the controls, with fewer complications. The conclusion of the author is that while sulfadiazine has a place in prophylaxis, its use is limited by the nature of the patient and of the infecting agent. Not much is gained by giving the drug before infection sets in. On the contrary, if sulfadiazine is promptly administered at the very beginning of an infection of the airways the result seems quite as satisfactory as if it were given in advance of an attack. While the author speaks of "no serious toxic effects" observed during his study, he says nothing of a possible sulfadiazine-fast condition, which would render this agent fairly useless when most desired.

VOORHEES, New York.

PARALYSIS OF THE LARYNX: AN EARLY SIGN OF RECURRENCE FOLLOWING RADICAL MASTECTOMY FOR CARCINOMA. J. R. FOX, Arch. Surg. **49**:388 (Dec.) 1944.

Hoarseness occurring after radical mastectory for carcinoma of the breast is often the first clinical evidence of metastasis. Metastasis of carcinoma of the

breast causes paralysis of the recurrent laryngeal nerve on the same or on the opposite side by involving the chain of lymph nodes surrounding the recurrent laryngeal nerve. Six illustrative cases are reported.

LIST, Ann Arbor, Mich. [ARCH. NEUROL. & PSYCHIAT.]

COLD STORAGE OF SKIN FOR AUTOGENOUS GRAFTING, FOREIGN LETTERS. D. N. MATHEWS, J. A. M. A. **128**:1041 (Aug. 4) 1945.

Skin preserved by cold storage can be used wherever grafting of skin is indicated. It makes the usual succession of operations unnecessary; the stored skin can be used without anesthesia as needed. Skin storage reduces the number of donor areas which are required in multiple stage plastic operations; it saves the patient the pain of repeated cuttings; the time required for each operation after the first is also lessened. Stored skin also provides an experimental skin bank for the study of heterogenous grafting. The author's method is as follows: The skin to be preserved is folded, with the under surfaces opposed, and then wrapped in a piece of *tulle-gras,* over which is placed a piece of gauze wrung out of isotonic solution of sodium chloride. The package is stored in a sterile, air-tight, screw-topped glass bottle and raised so that it will not touch the small amount of fluid which collects at the bottom. Storage was in an ordinary refrigerator at 3 to 6 C. This low temperature retards the autolytic activity of the tissue enzymes and the action of bacteria. Grafts of tissue which had been stored for periods up to twenty-one days took satisfactorily when applied to granulating surfaces. The theory of cold storage of skin for grafting was reported by Carrel in 1912 and by de Martigny in 1913, but Mathews' report is the first of stored skin being used in the treatment of burns in airplane accidents.

SELTZER, Philadelphia.

PENICILLIN IN INFECTIONS OF THE LUNGS AND BRONCHI. E. B. KAY and R. H. MEADE JR., J.A.M.A. **129**:200 (Sept. 15) 1945.

The authors indicate that penicillin is of great value in the treatment of acute pulmonary infections but that in the treatment of chronic pulmonary and bronchial infections its value is more limited. In general they concluded that the greater the degree of permanent destruction of tissue and infection, the less the benefit that was derived from penicillin. They comment in moderate detail on the bacterial flora in chronic pulmonary and bronchial infections and on the use of penicillin in combating bronchiectasis, chronic pulmonary abscess, suppurative pneumonitis, fungous infections and chronic bronchitis, they emphasize the value of administering penicillin intratracheally by indirect laryngoscopy and the use of a flexible laryngeal cannula (3 to 5 cc. of a saline solution of sodium, the salt of penicillin containing 250 oxford units to the cubic centimeter is used). The penicillin is injected at the end of expiration; this necessitates an inspiration before coughing is possible.

The intratracheal route seemed to give greater benefit. Many patients with advanced bronchiectasis received intratracheal administration of penicillin preoperatively. It was felt that intratracheal use of penicillin was of particular value in the treatment of chronic bronchitis, minimal bronchiectasis and advanced inoperable bronchiectasis and in preoperative preparation for lobectomy.

The chief value of penicillin in the treatment of pulmonary suppuration, chronic abscesses and fungous infections of the lung is its ability to decrease the general manifestations of toxicity, to promote symptomatic improvement and to prepare the patient for operative intervention.

GORDON, Philadelphia.

RECEIVING AREAS OF THE TACTILE, AUDITORY AND VISUAL SYSTEMS IN THE CEREBELLUM. RAY S. SNIDER and AVERILL STOWELL, J. Neurophysiol. **7**:331 (Nov.) 1944.

Snider and Stowell studied the electrical activity of the cerebellum in the cat and the monkey and the alterations in this activity produced by tactile, auditory

and visual stimuli. Distinct areas were found for the reception of impulses of each modality. Tactile impulses were received (*a*) in the lateral half of the anterior lobe and in adjacent folia from the ipsilateral half of the body and (*b*) in both paramedian lobules from the forefoot and the hindfoot of each side, the forefoot having a stronger contralateral representation than the hindfoot. In the anterior lobe and adjacent folia there was an anatomic localization with regional representation. The auditory area was restricted to the lobulus simplex and the tuber vermis. Decerebration failed to affect the auditory responses, but they were abolished by section of the eighth nerve and by destruction of the cochlea or the inferior colliculus. The visual area overlaps the auditory, and the pathways for the visual responses probably involve the superior colliculus. Full anesthetic doses of sodium pentobarbital did not affect tactile responses but depressed auditory and abolished visual responses. The authors suggest that cerebellar functions are influenced by tactile, auditory and visual stimuli, as well as by impulses from proprioceptors and from the cerebral cortex.

FORSTER, Philadelphia. [ARCH. NEUROL. & PSYCHIAT.]

THE RELATIONSHIP BETWEEN FREQUENCY AND DURATION OF MOMENTS OF STUTTERING. WENDELL JOHNSON and WILLIAM H. COLLEY, J. Speech Disorders **10**:35 (March) 1945.

Twenty stutterers (19 males and 1 female), ranging in age from 14 years, 6 months to 33 years, 6 months, with an average of 21 years, 1 month, read a phonetically edited 1,000 word passage under carefully controlled conditions. As the subjects read, the duration of each moment of stuttering was recorded on a tape by electromagnetic pens attached to noiseless signal keys manned by two observers working independently. On the basis of a low but positive relationship between frequency and duration of moments of stuttering, it is to be concluded that both frequency and duration of moments of stuttering are to be regarded as measures of severity or amount of stuttering, but neither is to be regarded as a complete measure since the two do not consistently correlate highly. Frequency alone or duration alone cannot be employed as a measure of the amount or the severity of stuttering. The severity of stuttering immediately following any one moment of stuttering cannot be predicted from the duration or the severity of the preceding moment of stuttering. The authors caution that any conclusions drawn must be evaluated in terms of the fact that the stuttering was measured in a reading situation only.

PALMER, Wichita, Kan.

DESCRIPTION OF THE PROFESSION OF SPEECH PATHOLOGY. AMERICAN SPEECH CORRECTION ASSOCIATION, J. Speech Disorders **10**:47 (March) 1945.

This article deals with the field of speech correction and, in general, the training and the function of the speech correctionist.

PALMER, Wichita, Kan.

THE INFLUENCE OF PROPOSITIONALITY ON STUTTERING. JON EISENSON and ESTHER HOROWITZ, J. Speech Disorders **10**:193, 1945.

Twenty-six stutterers, 23 males and 3 females, ranging in age from 17 to 20 years, with an average age of 18.4 years, read a word list of 130 words, a nonsense selection of 130 words and a meaningful paragraph of 130 words, the last taken from a letter written by the late President Roosevelt. Each selection contained the same 20 adjectives and the same number of parts of speech. Some duplication of words occurred, of course, in the three selections. (1) The increase in the propositional value of the oral reading selection resulted in an increase in stuttering on nouns, adverbs, verbs and adjectives. (2) The increase in propositional value of the oral reading selection resulted in a decrease in stuttering on pronouns, conjunctions and articles, with no significant difference with respect to prepositions.

(3) In general, nouns, verbs, adjectives and adverbs were stuttered more than pronouns, prepositions, conjunctions and articles. There is a greater difference in percentage of words stuttered between a meaningful and a nonsense selection than between a nonsense selection and the reading of a list of words. (5) An increase in propositional value produced a greater range of percentage of stuttered words among the various parts of speech. The authors conclude generally that the incidence of episodes of stuttering is directly related to the meaningfulness of the speech content. Stutterers have the great difficulty in the utterance of propositional speech.

PALMER, Wichita, Kan.

COMPLEXITY AND BREAKDOWN IN SPEECH SITUATIONS. CLARENCE T. SIMON, J. Speech Disorders **10**:199, 1945.

Simon presents a new test for integrative level in social situations to measure ability to handle situations of certain degrees of complexity. The test consists of laying playing cards, which have been arranged in a predetermined order, on a geometrically designed board so that like cards fall on like cards. This is done to the beat of a metronome every three seconds. As more and more cards are dealt, the situation gains in complexity. Students whose speech is normal but who were considered by their instructors to show marked stage fright tend to rate toward the lower end of the scale. Adult stutterers and stutterers from junior high school tend to rate lower on the scale than students with normal speech, and the differences are significant. Actual scores on college students range from 44 to 167, with 172 as a perfect score. Lower division class speech majors so far have a mean of 121. Upper class and graduate speech majors have a mean of 122. College stutterers have a mean of 103. Junior high school stutterers have a mean of 91. The author views stuttering as a breakdown of the total speech process, a disintegration of function. This breakdown may be occasioned by any or all factors which tend to increase the complexity of the speech situation beyond the stutterer's power to integrate. Conversely, any remedial approach which will simplify the speech situation or will reduce the complexity of the stimuli will tend to alleviate the difficulty.

PALMER, Wichita, Kan.

INTEGRATING THEORIES OF STUTTERING. Stanley Ainsworth, J. Speech Disorders **10**:205, 1945.

In a review of the literature Ainsworth states that modern theories of the cause of stuttering fall into three general groups: 1. Developmental theories, in which the stutterer is assumed not to be inherently psychologically or constitutionally different from the normal speaker but to have acquired stuttering speech in situations which occurred during his development. 2. Dysphemic theories, in which the stutterer is thought of as being somehow different from the nonstutterer, the exact nature of the difference not being thoroughly understood but probably occurring along biochemical, neurologic or physiologic lines. 3. Neurotic theories, in which the stutterer is assumed to be a psychoneurotic or neurotic person suffering from emotional maladjustments and presenting deviate behavior. This term as used here is intended to include milder states as well as fully developed neurasthenic and compulsive conditions, anxiety states and hysteria. Some theorists bridge one or more of these classifications.

PALMER, Wichita, Kan.

SPEECH INTELLIGIBILITY IN NAVAL AVIATION. M. D. STEER, J. Speech Disorders **10**:215, 1945.

In 1942 the Naval School of Aviation Medicine established a program designed to investigate the various aspects of voice communication in naval aviation. A modern laboratory was equipped and installed, which was capable of simulating the communication conditions and the transmission systems of flight. There are

four basic elements in all voice communications: (1) the text or language, (2) the speaker, (3) the transmission system and (4) the listener. Failure in any link of the chain will cause a breakdown in intelligibility of the communication. Research carried out by the experts employed by the United States Navy has attacked all of these issues.

PALMER, Wichita, Kan.

PRINCIPLES OF THE TREATMENT OF CHRONIC HEADACHE. ARNOLD P. FRIEDMAN and CHARLES BRENNER, New York State J. Med. **45**:1969 (Sept. 15) 1945.

The authors recall that "headache is a symptom, not a disease." It is associated, however, with a wide variety of diseases as dissimilar as tumor of the brain and chronic nephritis. Headaches are common in acute illnesses and disappear on convalescence, but long-standing recurrent headaches fall under three headings: (1) migraine; (2) psychogenic types; (3) post-traumatic types. Therapy, therefore, is dependent on pharmacology and psychology. The drugs used are of several classes: analgesics; sedatives; those acting on the autonomic nervous system; those acting on blood vessels; those affecting volume and composition of body fluids; and "miscellaneous" (hormones, vitamins, etc.). In patients with migraine, ergotamine gives the best results, administered orally as a tartrate in 1 mg. tablets; four or five are taken at first, then the tablets are taken at half hour intervals on a reduced scale. The post-traumatic types are treated with codeine, acetylsalicylic acid, or a short-acting barbiturate, but codeine is not to be used if the headaches are chronic. In all cases catharsis, desensitization, direction and reeducation are indicated. In "the vast majority of patients . . . psychologic factors are present . . . even though an underlying organic disturbance is present. . . . Unless the patient is enabled to cope with his psychologic problems, chances for a therapeutic success are minimal. . . ."

VOORHEES, New York.

EXPERIMENTAL PRODUCTION OF MOTION SICKNESS. E. A. SPIEGEL, M. J. OPPENHEIMER, G. C. HENNY and H. T. WYCIS, War Med. **6**:283 (Nov.) 1944.

The authors tested a number of medical students on a rotating-tilting machine. Symptoms of motion sickness were produced within eight minutes in 75 per cent of unselected subjects. The weaker method of stimulation (rotation combined with sagittal movements of the head or body) was sufficient to reveal a high degree of susceptibility. The stronger stimulation (rotation combined with frontal head movements preceding or succeeding rotation combined with sagittal head movements) was effective in nearly all moderately susceptible persons and produced symptoms even in some of the subjects who were supposedly not susceptible according to previous experience.

Optic fixation of an object that participates in all movements of the head tends to diminish the incidence of motion sickness as compared with watching of stationary objects of the surroundings.

Typical illusions of spatial orientation were produced. Their appearance was independent of that of the vegetative symptoms of motion sickness, indicating the presence of two rather independent mechanisms.

Symptoms of motion sickness may be elicited not only by stimulation of the maculas but by stimulation of the cristae ampullares.

PEARSON, Philadelphia. [ARCH. NEUROL. & PSYCHIAT.]

CHEMOTHERAPY OF INTRACRANIAL INFECTIONS: V. THE TREATMENT OF STAPHYLOCOCCIC AND PNEUMOCOCCIC MENINGITIS WITH SULFATHIAZOLE AND SULFADIAZINE. W. F. MEACHAM, E. SMITH and C. PILCHER, War Med. **6**:378 (Dec.) 1944.

The authors report the results of treating 152 experimental animals for staphylococcic and pneumococcic meningitis with sulfathiazole and sulfadiazine.

They found that sulfadiazine was present in the cerebrospinal fluid after oral administration in much greater concentration than was sulfathiazole. Experimental staphylococcic meningitis was not reduced by the oral use of sulfathiazole. The oral administration of sulfadiazine was distinctly beneficial in the treatment of dogs for staphylococcic meningitis. Experimental staphylococcic meningitis was not reduced by the intracisternal administration of suspensions of microcrystalline sulfadiazine. This form of therapy is believed to be harmful. The oral administration of sulfadiazine was decidedly beneficial in the treatment of dogs for pneumococcic meningitis. A high concentration of sulfadiazine in the cerebrospinal fluid was found to be essential to recovery in the experiments on dogs with pneumococcic meningitis.

PEARSON, Philadelphia. [ARCH. NEUROL. & PSYCHIAT.]

GRISEL'S DISEASE, OR NASOPHARYNGEAL TORTICOLLIS. L. EXPOSITO, A. DE FERIA and H. GONZALES, Bol. Soc. cubana de pediat. **15**:731 (Nov.) 1943.

This disease is characterized by acute torticollis, which produces permanent distortion.

Of sudden onset, it is always associated with an infectious process of the nasopharynx, followed by changes in the cervical portion of the spine due to rotation of the atlas in relation to the axis.

The history, a review of literature and a complete description of the illness, including the diagnosis and the pathogenesis, are given.

There is a summary in English of the clinical case presented.

KEITH, Rochester, Minn. [AM. J. DIS. CHILD.]

News and Comment

A COURSE IN AUDIOMETRY AND FITTING OF HEARING AIDS

The State University of Iowa, during the 1946 summer session, will offer as a feature of its full program in speech pathology and hearing conservation an intensive course in audiometry and the fitting of hearing aids. The course is designed to train the following groups: technicians to act as lay assistants to otologists; school nurses to test the hearing of children of the public schools; executive secretaries of leagues for the hard of hearing, and others interested in the conservation of hearing. Any person who meets the college entrance requirements is eligible. The 24 students whose applications are received first will be accepted for the course. The course will run from June 17 to July 29. Applications may be made to Prof. Wendell Johnson, Director of the Speech Clinic, University of Iowa, Iowa City, Iowa.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION

The American Broncho-Esophagological Association will hold its annual meeting in Chicago, May 31, 1946, at the Drake Hotel.

Directory of Otolaryngologic Societies *

INTERNATIONAL

First Pan-American Congress of Oto-Rhino-Laryngology and Bronchoesophagology

Predident: Dr. Gordon B. New, Mayo Clinic, Rochester, Minn.
Secretary: Dr. Chevalier L. Jackson, 255 S. 17th St., Philadelphia 3.
Place: Palmer House, Chicago. Time: Oct. 17-19, 1946.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia.
Secretary: Dr. Fletcher D. Woodward, 104 E. Market St., Charlottesville, Va.
Place: San Francisco. Time: July 1-5, 1946.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Gordon B. New, Mayo Clinic, Rochester, Minn.
President-Elect: Dr. Alan C. Woods, Johns Hopkins Hospital, Baltimore 5.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100–1st Ave. Bldg., Rochester, Minn.

American Broncho-Esophagological Association

President: Dr. Carlos E. Pitkin, Carnegie Medical Bldg., Cleveland.
Secretary: Dr. Paul Holinger, 700 N. Michigan Ave., Chicago 11.
Place: Drake Hotel, Chicago. Time: May 31, 1946.

American Laryngological Association

President: Dr. Harold I. Lillie, Mayo Clinic, Rochester, Minn.
Secretary: Dr. Arthur W. Proetz, Beaumont Bldg., St. Louis.
Place: Drake Hotel, Chicago. Time: May 29-30, 1946.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Albert C. Furstenberg, University Hospital, Ann Arbor, Mich.
Secretary. Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.
Place: Drake Hotel, Chicago. Time: May 27-28, 1946.

SECTIONS:

Eastern.—Chairman: Dr. Philip E. Meltzer, 20 Charlesgate West, Boston 15.
Southern.—Chairman: Dr. E. Trible Gatewood, Professional Bldg., Richmond 19, Va.
Middle.—Chairman: Dr. Lawrence R. Boies, Medical Arts Bldg., Minneapolis 2.
Western.—Chairman: Dr. Clarence A. Veasey Jr., Paulsen Bldg., Spokane 8, Wash.

American Otological Society

President: Dr. Gordon Berry, 36 Pleasant St., Worcester, Mass.
Secretary: Dr. Gordon D. Hoople, 713 E. Genesee St., Syracuse, N. Y.
Time: May 31-June 1, 1946.

American Otorhinologic Society for the Advancement of Plastic and Reconstructive Surgery, Inc.

President: Dr. Alfred Schattner, 115 E. 61st St., New York.
Secretary: Dr. Norman N. Smith, 291 Whitney Ave., New Haven, Conn.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.